

HÆMORRHOIDS AND THEIR TREATMENT:

THE VARICOSE SYNDROME OF THE RECTUM

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AUTHOR'S PREFACE TO THE ENGLISH EDITION

SINCE the appearance of the German edition of this book in October, 1935, I have treated a further 1100 new cases of anorectal disease, as well as giving further series of treatment to just over 300 patients who had already had treatment by me. Some of these latter had very long-standing disease, and a number have now had three to four series of injections. All this fresh material is now included in the English edition, it includes 80 new fistula cases, 200 new fissures, and 180 new cases of pruritus ani.

In none of these cases was any serious complication, attributable to the treatment, observed. This may possibly be due to the fact that in the last three years I have both lowered the concentration of the injection solution and diminished the amount used at a time. The first treatment is now limited, almost without exception, to three injections of 3 minims each (about a third of the quantity formerly used) given with the Blond drop-syringe. Every patient is digitally examined before treatment is begun, in order to eliminate the risk of giving the injection in the presence of commencing inflammation in a thrombosed varix. If the examination is painful, or if marked sensitivity suggests the possible presence of such an infection, treatment is withheld for a few days until it is either revealed or excluded. Where, however, as is much more commonly the case, such tenderness is due to a hidden fissure, the latter may be dealt with at once, and the treatment of the internal varices begun after an interval of five to eight days.

In addition to my own personal cases, treated without mishap, some six to seven hundred cases which have now been treated by my pupils in various places should be mentioned. Without doubt the real total of such cases must be far greater, for many of my pupils have not as yet published their results. Among those who are now practising the technique which they learnt from me are Baumann in Zürich, Bohm, Samet, Haber, and Lewith, in Prague, Kassowitz

and Lukic in Jugoslavia, Cholkar in India, Lopez Gonzalo in Spain, Lehnert and Wilde in Germany, O'Loughlen in London, Papier in Poland, Parker in Boston, Perriera in Portugal, and Szandor in Hungary. Quite a number of institutions in Austria and Germany have now adopted the methods.

Whereas in Austria and Germany, at least in the early days of my injection treatment, a great deal of opposition had to be surmounted, in England and America the method needs no apology, being already established and having the approval of leading proctologists. Of relatively minor importance is the actual solution used for injection. Every experienced worker has his own well tried preparation, his own instruments, and a technique to which he is accustomed. On the other hand, it does seem to me important that preference should be given where possible to methods which are ambulatory and which do not in the least interfere with a patient's ability to continue his work. For this reason I hope that my advocacy of injection treatment for fissure, fistula, and pruritus will prove acceptable in the English-speaking countries.

In the injection treatment of rectal varicose disease it is the *underlying theoretical principles* which have hitherto received least attention. This defect I have endeavoured to remedy, and it is these basic principles, together with the hypotheses which spring from them, that I am anxious to bring to the notice of the medical profession in England and America. In particular, I hope that the intimate relation between anorectal varicose disease and the portal-caval circulation will now receive the notice it deserves.

The German edition was published with the assistance of my pupil, Dr Herbert Hoff. For the content of the book I alone am responsible, most of the material having been published beforehand in a number of papers. To Dr Hoff my sincere thanks are due for the compilation of the statistics and for the management of the notes of the cases (700) seen at Prof. Schonbauer's clinic in Vienna. The remainder (2400) were seen by me in private. All the 3100 cases were treated by me personally.

KASPER BLOND

TRANSLATOR'S PREFACE

SOME sections of this book, such as those on the injection treatment of hæmorrhoids, will cause no surprise or comment in England. Others, for example on the ætiology and treatment of fistula and pruritus, will be new to many readers. Others again, and in particular the theoretical concepts of the portal system and its role in the causation of varicose disease, will be not only new but startling. It is hoped that their wider discussion will stimulate experimental and clinical work and thus help to verify and elaborate them.

This translation has been prepared in close collaboration with the author, who has added his more recent data and experiences and made other slight modifications of the original German. A list of references has been appended.

Both Dr. Blond and I desire to place on record our appreciation of the very great care, skill, and willing co-operation of Messrs John Wright and Sons.

E S I

CONTENTS

| CHAPTER | PAGE |
|---|------|
| PREFACE TO THE ENGLISH EDITION | iii |
| TRANSLATOR'S PREFACE | v |
| I —THE AETIOLOGY OF HÆMORRHOIDS | 1 |
| II —SYMPTOMATOLOGY | 6 |
| III —DIAGNOSIS AND INVESTIGATION | 11 |
| IV —THERAPY | 16 |
| V —TECHNIQUE OF TREATMENT | 25 |
| VI —ANAL FISSURE | 38 |
| VII —CONSTIPATION | 51 |
| VIII —SPONTANEOUS THROMBOSIS IN THE RECTAL VEINS—LOCAL AND GENERAL PHLEBITIS—THE PROBLEM OF EMBOLISM | 54 |
| IX —ACUTE AND CHRONIC SUPPURATIVE THROMBOPHLEBITIS OF THE RECTUM AND ANUS—PROCTITIS AND FISTULA IN ANO | 78 |
| X —TREATMENT OF FISTULA | 99 |
| XI —THE ROLE OF THROMBOPHLEBITIS OF THE HÆMORRHOIDAL VEINS IN THE AETIOLOGY OF BILIARY DISEASE | 117 |
| XII —ANORECTAL MUCOSAL PROLAPSE | 121 |
| XIII —VARICOSE VEINS OF THE LEG IN RELATION TO HÆMORRHOIDS | 126 |
| XIV —INSTRUMENTARIUM | 129 |
| REFERENCES AND BIBLIOGRAPHY | 134 |
| INDEX | 138 |

HÆMORRHOIDS AND THEIR TREATMENT

CHAPTER I

THE AETIOLOGY OF HÆMORRHOIDS

OUR ideas and theories regarding the origin, pathology, symptomatology, and treatment of hæmorrhoids have for long stood in need of revision. There is, for example, no consensus of opinion as to whether 'hæmorrhoids' is a disease *sui generis*, or whether the dilated veins are merely symptoms of some systemic disorder. The typical hæmorrhoid, consisting of a cluster of small prominent veins, and tending to prolapse downwards to some extent on defæcation, is responsible for an important group of symptoms. Yet it is probably not correct to regard these dilated veins as in themselves constituting a disease, for they depend upon some venous congestion for their formation, and for this congestion in turn there must be some underlying cause, such as, in some instances, a dysfunction of the liver.

Any one examining many cases of hæmorrhoids will notice certain variations which are physiological rather than anatomical. Thus, in the same patient there are changes in the tone of the sphincter ani, which may vary from a state of hypertonus to one of hypotonus. Also a very great difference in the degree of prominence of the hæmorrhoids may be noticed from time to time, it will differ before and after defæcation, and the type and quantity of food ingested and the stage of digestion will also produce variations. Such changes can readily be made out with the proctoscope.

The term 'hæmorrhoid' is usually understood to mean an anorectal varix. Such varices are commonly subdivided into *external* and *internal*. But taken literally, the term connotes bleeding. Now there are many patients with anorectal varices who are never troubled with bleeding, and these individuals may be quite unaware that

hæmorrhoids are present. Yet a variety of manifestations other than bleeding may occur, for which the 'hæmorrhoids' are responsible, so that the designation appears to the author not particularly apt, and in some cases misleading. Similarly the term 'pile' (*pila*, a ball), referring as it does to palpable swellings, is vague and unsatisfactory. In this book we shall bring together under the general heading "*varicose syndrome of the rectum*" a group of manifestations, often hitherto regarded as independent entities, and we shall try to show that they are bound up with the presence of rectal varices. These manifestations are *erythema, eczema, and pruritus ani, pruritus vulvæ and scroti, anorectal thromboses (internal and external), anal fissure and stercoral ulcer, proctitis, peri-anal infection, and anal fistula, and prolapse of the rectal mucosa*. We confine the term 'hæmorrhoids' to the actual mucosal prominence due to the underlying varix, and for the firmer swellings due to thrombosed varices we use the Latin word 'nodulus'. No doubt the latter term was originally introduced merely to emphasize that patients with rectal varices were prone to the sudden appearance of nodes or swellings about the anus. Nowadays, however, there is a tendency to forget that the noduli are merely symptoms and to regard them as a separate complaint. Thus many people consider these external thromboses to be inflammatory in origin, but this is not the case. Inflammation may certainly follow as an accidental complication, but it is by no means the rule. Another common error is to mistake such thrombosed external varices for prolapsed 'internal piles', and to attempt their replacement. The true nature of these noduli will be considered fully in a later chapter, but this preliminary critical examination of the terminology is essential, because the loose use of terms such as the above tends to perpetuate errors though it may serve to cover our ignorance. The old-fashioned terms 'internal' and 'external' hæmorrhoids should be abandoned because in reality there are no such things as external hæmorrhoids. There are indeed varices under the anal skin belonging to the inferior hæmorrhoidal plexus but they scarcely ever bleed. The so-called 'skin-tags'—generally quite harmless—upon the removal of which operators place such stress, are merely unimportant indications of the underlying varicose condition. The redundant skin-fold may distend with blood during defæcation, but is scarcely ever a source of bleeding. The characteristic and sometimes alarming hæmorrhages to which these patients are liable always come from internal varices. A diagnosis of external

bleeding is generally due to mistaking a prolapsed, strangulated, internal varix for an external

This bleeding may serve as a convenient starting-point for the argument of this book. In more than one instance, patients of ours have given a story of having been found fainting while at stool, having lost a pint or more of blood quite suddenly. When such patients are examined proctoscopically, it is astonishing to find that the varicose vessels appear to be quite small. The source of the bleeding can as a rule be seen easily, and if the small covering thrombus is removed, blood only trickles out drop by drop from the vein. The question therefore arises, how it is that during defæcation such terrifying bleeding can occur from these same small vessels.

The answer is that blood is forced backwards from the valveless portal system. During proctoscopic examination the blood-flow is upwards, i.e., hepatopetally, and only escapes in a small trickle from the vein, but during defæcation there is in these same patients a reversal of flow in the portal circulation, exactly comparable to that which occurs in a saphena varix. This, in the case of the saphenous system, may be demonstrated by what is called Trendelenburg's sign. *This temporary reversal of flow in the portal circulation is the true cause of anorectal varices.* All pathological conditions which impede the portal venous return may thus play a part in the development of hæmorrhoids. Examples are heart diseases, cirrhosis and inflammatory swelling of the liver, and hepatic metastases. Moreover, diseases of organs adjacent to the portal vein, inasmuch as they may produce pressure on the vein, may be contributory factors. Examples again are stone in the common bile duct, and tumours of the pancreas or the right kidney.

Thus it will be seen that the rectal varicose syndrome is not to be regarded as an idiopathic disease. This was realized quite well long ago, as the following quotation from Wunderlich (1856) on the subject of piles shows —

In a disease which commences insidiously, and which may be well established before definite symptoms occur, it is difficult to trace out the original causes. Nevertheless it is generally accepted that any condition which leads to congestion of the pelvic viscera by interfering with their venous return, will favour the development of hæmorrhoids. This manifestation of the pelvic congestion may in some cases be predominant, and the underlying causes unobtrusive and non progressive. In other cases, however, some important and serious disease of the heart, liver, or bowel may be present, though overshadowed by the rectal symptoms.

A second quotation from the same author will serve to show how our ideas have advanced with the increase in our knowledge of cellular pathology —

The diagnosis of hæmorrhoids may often be difficult. On the one hand it does not follow that rectal bleeding, external noduli, and other manifestations are purely local in origin. They may well be mere by-products of some other disease as of the heart or liver. On the other hand, the absence of obvious pathological changes at the anus does not exclude the existence of hæmorrhoids. In many cases after long observation the diagnosis of hæmorrhoids is finally established by a rectal hæmorrhage. Hence it is not surprising that diagnostic mistakes in both directions are of daily occurrence.

And whereas in the past the tendency was to attribute symptoms of the most widely different origins to piles, so that important systemic diseases were not infrequently overlooked, the inclination nowadays is to err in the opposite direction, and to misunderstand many clinical pictures by not giving due weight to the presence of piles and their implications.

That the rectal varicose syndrome occurs mainly in adults is to be expected, for the diseases which cause it are chiefly those of adults. It is not an inherited disease or diathesis as is widely taught, for if it were, we should see the development of hæmorrhoids in children as commonly as in adults. The condition is rather to be regarded as a wearing-out process. Although a few cases of juvenile or adolescent hæmorrhoids have come to our notice, a definite exogenous cause could be found in all these cases. The higher the average age of a given group of people, the greater will be the incidence of rectal varicose disease, in other words, the incidence is that of a 'wear and tear' disease. In apparently healthy individuals, so-called idiopathic hæmorrhoids may generally be shown to owe their origin in adults to overeating and in children to faulty habits of feeding etc. The abuse of aperients would seem to be one common ætiological factor. The hæmorrhoidal patient is often of plethoric build, with prominent abdomen. The result of overeating is that the liver is chronically overloaded.

Some have supposed that there exists a racial predisposition to piles, but this would probably be explained rather by differences in food and the methods used to prepare it—some methods quite conceivably being more harmful to the liver than others. Children of all races are alike practically exempt from the disease as long as they are properly and naturally fed. In all races alike piles are a complaint of later life, when faulty habits of eating and drinking

have become established. In the author's series of more than 3100 cases no definite evidence of any racial predisposition could be found.

Two important predisposing causes are often considered to be a sedentary life and chronic constipation. Yet a sedentary life is by no means an essential factor, for we have seen large piles in young professional footballers and other athletes whose mode of life was obviously a very active one. As for constipation, we shall later bring forward evidence that this is not a cause, but merely another symptom of the varicose syndrome. Many hæmorrhoidal patients will be found on careful questioning and examination to suffer from stomach, bowel, liver, or biliary disorders, and to this interesting fact we shall refer later.

Supporters of the 'inflammatory theory', among whom Quenu and Hartmann are prominent, base their opinions largely on the inflammatory changes which may be demonstrated histologically in the varicose anorectal veins. Our contention is that these changes are secondary in nature. Uncomplicated rectal varices are rarely if ever infected; infection supervenes only after thrombosis has occurred. Even then, infection is by no means the rule, but rather the exception.

CHAPTER II

SYMPTOMATOLOGY

I EXTERNAL VARICES

IT is possible to distinguish three kinds of anorectal varices. *Large calibre veins* are to be found under the mucosa 5 to 8 cm. above the anus, and these, which belong to the superior hæmorrhoidal plexus, perforate the muscular coat and *anastomose with veins of the genital plexuses*. Lower down are *medium sized veins*, mostly belonging to the middle hæmorrhoidal plexus, and in the region of the external sphincter are *small veins* constituting the inferior hæmorrhoidal plexus. These latter varices are dilatations of quite minute vessels.

This classification is in our opinion more valuable than the usual ones. It affords a useful comparison with varicose veins of the lower extremity. Here the largest varices are almost entirely confined to the thigh, they hardly ever extend below the knee. Smaller veins are found chiefly on the dorsum of the foot and over the malleoli. The minute dilated veins—telangiectases—are however seen at all levels and are a certain sign of venous stasis. Varices in the ampullary part of the rectum are rare, they have been described in the literature, but are not of practical importance. *Surprisingly often anorectal varices will be found to coexist with leg varices, and with varicocele*. Varices at the anal margin may exist for years without causing bleeding, pain, or any other symptom. They may, however, be detected by a physical sign which has not hitherto been described in the literature. If a patient with such varices is told to 'bear down', the veins become distended with blood and are then visible as a blue discoloration showing through the thin anal skin.

These small varices are very commonly met with near the mucocutaneous junction, and in this situation sudden spontaneous thromboses are prone to occur. There is often a history of diarrhœa, or of the use of some strong purgative, just before this happens. The thrombosis is very painful, and the patient is unable to sit down comfortably. There may be severe tenesmus, and a feeling as of a foreign body in the rectum. Sometimes a localized swelling may be seen, beneath which a little group of such thromboses is to be

felt. The patients are often alarmed by the severe pain, which interferes with their sleep, and soon compels them to seek advice. Very often a wrong diagnosis of 'strangulated piles' will be made, and hot applications and the traditional sitz baths ordered, or the doctor may waste his time and trouble in efforts to 'replace' the 'prolapsed' pile.

If such a spontaneous thrombosis is left to take its course, after about eight days (even without poultices, aperients, and sitz baths) the blue colour fades, the previously somewhat œdematous nodule becomes shrunken, the contained clot is absorbed, and the end-result is an ordinary 'hypertrophic skin-tag'. In other cases a painful process of ulceration occurs, and a small hole appears in the thin anal skin. This gradually enlarges, and finally the clot breaks through and is extruded. In other cases again, there may be great pain with fever, and suppuration occurs in the clot before it ulcerates through. In this way a little abscess is secondarily formed, which either bursts on to the surface or has to be incised. If such a thrombus is situated at the mucocutaneous junction, especially at the posterior or anterior commissure, but rarely to right or left, it may, after the loss of the tiny infected thrombus, develop into a *typical anal fissure*.

We may correlate these various clinical pictures by stating that they are several stages of the same process. First of all, we must distinguish a stage in which the varices produce few or no subjective symptoms, yet may be seen shining through the anal skin when they are distended. The blood which distends them as the patient is made to 'strain down' comes from the portal system, and rapidly drains off into the caval system when the pressure is released. Yet although this intermittent reversal of flow is already present, it will be noted that the patient has no symptoms. In the second stage, spontaneous thromboses appear with swelling and severe pain, but with no evidence of inflammation. In these cases, incision of the thrombosed nodule and evacuation of the clot will usually result in healing *per primam* within three or four days. If untreated, the patient enters the final stage. This is characterized as described above by the spontaneous extrusion of the clot without infection, or, more rarely, by infection of the clot *in situ*, the clot being then extruded by a process of suppuration. At the anal margin this stage of acute infection constitutes what is generally known as a 'peri-anal abscess'. It would, however, be more accurately styled

'acute anal thrombophlebitis' If, alternatively, the thrombosis is a little above the anus, near the mucocutaneous junction, its epithelial covering is much thinner, and rupture occurs without severe symptoms, the process being subacute. The superficial wall of the nodule necroses, and the tiny clot falls away or is separated by slow suppuration. Thus is formed the 'anal fissure' which would be much more accurately called '*chronic anal thrombophlebitis*'

II INTERNAL VARICES, OR HÆMORRHOIDS

The present medical generation has not given to so called 'internal piles' the attention which they merit. The early stages are considered unimportant, and only the relatively late stages of bleeding or prolapse are thought worthy of treatment. And yet internal varices are of much greater clinical importance than external, their manifestations are much more complex, and their early treatment may serve to prevent a train of serious late complications. It is well recognized that varices in the zone of the middle and superior hæmorrhoidal plexuses may run a long latent course before the first symptom makes its appearance. This latency, however, is more apparent than real. It is true that during this stage the classical signs of piles—namely, prolapse and bleeding on defæcation—are absent. Nevertheless symptoms are present, but of a kind not hitherto recognized as being hæmorrhoidal in origin. Many patients with internal varices have, for example, chronic constipation, others suffer from a serous discharge with a constant disagreeable moistness of the anal region, which may later pass on to erythema, eczema, or pruritus ani. In most patients at this early stage there will be found no sign of peri-anal skin tags, on the contrary, the anus often appears smoother than the average. Only one sign is present which will reveal to the experienced clinician that he is dealing with a case of rectal varicose disease—on digital examination a quite definitely increased resistance of the sphincter will be apparent. There is in fact a distinct hypertonus of the sphincter, and these patients tend to be particularly sensitive to both digital and instrumental examination.

The subjective symptoms of internal varices are very varied. One patient, for example, will complain of chronic persistent discomfort, perhaps of pain in the back, while another will have periods of complete well-being with attacks of pain or bleeding in between. Many patients have trouble only during defæcation, when prolapse

of swollen mucosa tends to occur. Patients soon learn how to replace the prolapse, the swelling is tender to touch, but on the whole the pain is bearable, generally no more than a burning sensation. As time goes on, these patients are distressed by constant soiling of their clothes, and increasing difficulty in keeping themselves clean, some develop quite an elaborate ritual with this object. This stage may continue for many years, possibly even ten or twenty, and is characterized by a hypotonus and atrophy of the sphincters. In course of time a sneeze or cough is enough to bring down the prolapse. These patients can then no longer trust themselves in the street or in company, for the prolapse demands immediate replacement. The manner of the bleeding may also be characteristic, it may be very slight, perhaps appearing as bloody streaks on the surface of the stool, or it may come at the end of defæcation in a little spurt.

It is a common belief that strong expulsive efforts may cause a hard faecal mass to damage superficially placed varices, but this conception, as already pointed out, is unnecessary and erroneous. If these patients with daily bleeding are examined proctoscopically, even before defæcation it will be observed that, though the rectal ampulla is empty there is a small trickle of blood oozing from congested varices. These same patients at stool may in a few moments lose half a pint or more of blood. This blood is, surprisingly, not dark but bright, and spurts out forcibly. It is this striking fact which caused Allingham to postulate the existence of arterial varices. Its true significance may, however, be made clear by the following clinical observation. If a moderately large vein is pricked or injured dark blood escapes from it drop by drop. If now the venous return is obstructed, bright blood will spurt from the vein. Something similar may be observed in the large varicose veins of the lower limb. If such a vein in a patient with a positive Trendelenburg's sign (that is, a reversed blood-flow in the saphenous system) ruptures, blood gushes out in a jet. Under normal conditions, and during proctoscopy, the blood in the hæmorrhoidal veins is flowing upwards (hepatopetally), but while straining at stool the stream is reversed, and the flow is downwards to the anus (hepatofugally). The bright blood which the patient with hæmorrhoids loses in a sudden rapid gush is actually coming from the portal system. The great sense of relief and well-being which a patient will sometimes feel after such a bleeding may be explained

as due to the elimination of toxins which were carried in the portal blood. The older physicians knew that a hæmorrhage of this sort might often be beneficial —

Bleeding, as long as it is moderate, should not be interfered with. All that is needed is a careful diet, moderate exercise and the avoidance of local or general irritation. Only when bleeding is very prolonged or severe or if the patient is already very weak, is intervention necessary (Wunderlich).

Most hæmorrhoid patients suffer from abdominal plethora, and their liver metabolism is usually overcharged. It may be that these bleedings act as a safety-valve to prevent damage to the liver.

Not all these plethoric patients are constipated, some tend to suffer from diarrhœa. The author interprets the onset of this diarrhœa as a sign of a greater degree of liver damage. It occurs only when portal congestion has reached an advanced stage. In many sufferers from hæmorrhoids, imperfect liver function and dyspepsia are prominent. Sometimes the whole gastro-intestinal tract is secondarily affected by the portal congestion. The possibility that gastritis and duodenitis may be but symptoms of portal congestion cannot be lightly dismissed. As we have already indicated, the older generation, who were less prone to think of diseases as separate and local entities, accepted it as self-evident that gastro-intestinal disturbances should go hand in hand with hæmorrhoidal symptoms. The author's own experiences have led him to believe that in this the last generation was nearer to the truth than the present. The rectal varicose syndrome is not a localized disease of one organ, but rather an indication of a dysfunction of the whole alimentary tract.

CHAPTER III

DIAGNOSIS AND INVESTIGATION

THE investigation of a patient with rectal disease is not such an elementary matter as is generally supposed. It consists of more than digital and endoscopic examination. It commences with the taking of a careful history, on which the subsequent procedure is based. The methods to be adopted depend upon the patient's presenting symptoms. It must be remembered that the slightest error of judgement in dealing with these nervous and anxious patients may lead to a refusal of further treatment. For example, an attempt at digital examination in the presence of a fissure may be so painful that the patient's confidence is lost. It must also be borne in mind that patients who have been operated on one or more times for hæmorrhoids will most likely have a stenosis or partial stenosis of the anus which contra-indicates any attempt at either digital or instrumental examination without preliminary local anæsthesia.

It is important to obtain a clear idea of the type of pain complained of. Constant burning pain suggests internal hæmorrhoids. Pain coming on in attacks which begin immediately after defæcation, remain for some time, and slowly pass off, to return again similarly at the next defæcation, suggests an anal fissure. Pain appearing suddenly in a previously healthy individual, persisting day and night, and independent of defæcation, indicates a spontaneous peri-anal thrombosis. Constant pain with fever should make one think of an infected thrombus, i.e., an acute anal thrombophlebitis (so-called periproctitis). The character of the bleeding, also, may be an important aid to diagnosis. The occasional appearance of streaks of blood on the fæces or the toilet paper suggests a fissure. A history of constant soiling of the linen with blood indicates that some degree of prolapse is present. If the blood is watery and mixed with pus, proctitis or carcinoma must be thought of.

The sudden appearance of a quantity of pus is usually due to spontaneous rupture of an abscess. Where chronic suppuration has followed an operation, a fistula may be suspected. Slight purulent discharge with pain may be due to either fissure or fistula.

There are important combinations of these symptoms, for example, a purulent discharge with a history of prolapse suggests necrosis of irreducible prolapsed internal piles. Another presenting symptom is peri-anal itching and moistness, most marked at night. This itching may spread from the anus to the scrotum or vulva and the groins.

When investigating patients with rectal varicose disease, one should on principle inquire for any evidence of gastro-intestinal trouble, and especially as to previous liver or biliary disease. Urinary and prostatic symptoms must also be sought for because, as we shall show, these organs may sometimes become affected.

The next step in the investigation is inspection. This alone will sometimes suffice to confirm the diagnosis foreshadowed by the history. Patients with spontaneous thrombosis, fissure, fistula, or peri-anal abscess will often not permit anything more than inspection. For fissure, local anæsthesia will make digital examination possible, a thrombosis must first be anæsthetized and evacuated.

When a thorough external examination has been made, and any external varices noted, digital examination may be attempted provided that the above contra indications have been dealt with. The surgeon must not expect to detect internal varices at this stage. hæmorrhoids cannot be felt, they must be looked for. Palpable lesions are thromboses, ulcers, polyps, infiltration, scars and fibrous bands, and the results of injection treatment. Instrumental examination must next be undertaken. Rectoscopic findings at the higher levels will be found fully described in text-books and reference works. We are here solely concerned with the lowermost few inches of the bowel. Large varices seldom extend to the upper part of the ampullary portion, so that for the study of these lesions the Blond proctoscope with lateral window is quite adequate (*see p 130*). But before confining one's attention to the zone visible through this instrument it is important to exclude any lesion at a higher level.

It must here be emphasized that many of the proctoscopes in general use are quite unsuitable for the thorough investigation of anorectal disease. The more accurate understanding of rectal varicose lesions has been made possible by the introduction of the proctoscope with the side window.

From time to time it will be found that a patient attending with piles has a cancer of the rectum, often still operable. Moreover, cancer of the sigmoid colon may at a relatively late stage lead to the development of large rectal varices. In this case the infiltration of

the tumour and its increasing size lead to a congestion in the superior hæmorrhoidal vessels. In one of our cases we were able to diagnose, on the evidence of a ring of high lying thromboses liver metastases which were shortly afterwards verified at autopsy.

Of the greatest importance is the correct interpretation of the proctoscopic findings and the recognition of the appearances seen in varicose disease. It is very common to find doctors who are considered to be experts in the subject misinterpreting these findings



Fig 1.—Proctoscopic appearance of ordinary varix



Fig 2. Proctoscopic appearance of a collection of small varices



Fig 3. Proctoscopic appearance of a red un-sized varix



Fig 4. So-called hæmorrhoidal papilla toppling a varix

The patients are examined with the ordinary proctoscope and the doctor expects to find varices resembling those of the lower limb. As he cannot see them he concludes that no piles are present. Neither the absence of skin tags nor the lack of a history of bleeding is any indication that internal varices are not present. It is in fact very difficult to diagnose hæmorrhoids with the ordinary proctoscope—the passage of the instrument flattens and empties the varices. The illustrations (Figs 1-4) give a better idea than any verbal

description of what must be looked for. Only long experience, both of normal and abnormal rectums, will enable one to recognize all the various pathological appearances.

The analogy with the lower limb may be of some assistance. The large calibre varices of the rectum resemble the large varicose veins of the thigh and upper third of the leg; the medium-size varices are like those on the leg, particularly of the external saphenous group; the smaller varices resemble those on the dorsum of the foot and over the malleoli, while the minute dilated venules seen especially in proctitis are like the telangiectases seen on the legs of women after childbirth.

The normal colour of the rectal mucosa is rose-pink, and in the healthy rectum the mucosa protrudes hardly at all into the lumen



Fig. 5.—Proctoscopic appearance of large varix with dilated venules.

of the proctoscope. The diseased mucosa is dusky red, or even bluish black, and has a rough, granular, almost œdematous-looking surface. By using the lens of the proctoscope, one can clearly make out minute vesicles and tiny erosions where the epithelium is deficient. The condition of the mucosa may be compared with that of the skin in peri-anal eczema; in both places there are little localized excoriations of the epithelium, with punctate hæmorrhages, and in both places there is a constant sero-sanious exudation. The larger varices

may be more deeply, so as to be obscured by the thick velvety mucosa, they then only betray their presence by prolapsing into the lumen of the proctoscope. At times, the mucosa may appear inflamed, and fiery red. Large superficial varices will show bluish black through the mucosa, and on their surface may clearly be seen dilated vasa vasorum (Fig. 5). Sometimes ulcerated areas are clearly visible against the dark vermilion background. Sometimes, too, a hæmorrhoid may be seen topped by a so-called 'hæmorrhoidal papilla'—a tooth-like, polypoid, greyish-white structure (see Fig. 4). Another interesting observation is that large polypi, when present, tend to be situated in the hæmorrhoidal zone.

Such are the changes which are responsible for the main symptoms of hæmorrhoids—the pain or burning on defæcation, the sense of fullness in the rectum, the moistness and irritation. As we

have already pointed out, there is nothing in these changes to suggest that they are primarily infective in origin. In our opinion they are a direct result of the damming back of portal blood overladen with metabolites and other poisonous substances. Often a sharply demarcated dark zone may be seen standing out clearly against the neighbouring brighter mucosa, and looking almost as if it had been painted. This is the boundary between the middle and inferior hæmorrhoidal plexuses.

When we succeed in discovering all the physiological or pathological conditions which may lead to a reversal of blood-flow in the portal system, either intermittent or permanent, the ætiology of rectal varicose disease will become clear. We shall also very probably attain to a better understanding of many metabolic disturbances, especially of the liver.

A very disagreeable symptom of the varicose syndrome is the intermittent prolapse of the mucosa. At first the patient is able to replace the prolapse, but in course of time the increasing discharge and constant soiling of the clothes makes life a misery for these patients. We treated one patient a colleague, who for years had avoided going out in the afternoons because if he even sneezed or coughed the prolapse would occur, and need immediate replacement. True irreducibility is uncommon, so called incarceration only occurring if replacement is delayed. Then the commencement of inflammatory changes and necrosis lead to œdema of the anal ring, and the patient fears to make any attempt at reduction on account of the pain.

It has already been pointed out how commonly this condition is confused by doctors with thrombosis of external varices. External thromboses are of course irreducible, the proper treatment is immediate incision. Prolapsed piles and mucosal prolapse are almost always replaceable if properly handled, so-called incarceration is generally the result of wrong management. The picture in such cases is a pathetic one: there is intense pain and constant tenesmus, making rest impossible, fever may be present. The author has seen a number of patients whose prolapses were still reducible in spite of weeks of wrong treatment by bed, hot compresses, sitz baths, etc. Even at this late stage complete relief may be secured by correct and energetic treatment.

CHAPTER IV

THERAPY

THE ancients were well acquainted with the operative treatment of piles. Hippocrates in 400 B.C. was already practising operation by ligature and puncture. Celsus in Rome at the time of Tiberius performed excision. In Spain Albucasis, the Arab physician, was accustomed to treat piles with the cautery. The first to use injection methods appear to have been certain English charlatans known as "pile-curers". Their methods and solutions were secret, but as early as 1853 Jobert had learned and adopted their treatment.

In 1869 the method was extensively used by Morgan, a Dublin surgeon, who employed a ferric sulphate solution, and reported good results. In 1871 Mitchell of Illinois used phenol solutions for the same purpose. He and his school advocated a 50 per cent solution, the object being to cause sloughing of the treated piles. Nowadays a 5 per cent solution, in oil or in glycerin, is popular, the aim being not to cause sloughing but to sclerose the varices by causing inflammatory fibrosis in the submucosa.

In 1913 E. H. Terrell, of Richmond, U.S.A., introduced the 5 per cent quinine urea solution. After the war, Boas commenced to use the method in Germany. He employed a 70 per cent solution of alcohol in conjunction with the suction method of Strauss, in which the piles were drawn outside the anus for treatment by the application of a suction glass. It is impossible to mention in detail here all the various solutions, their concentrations and combinations which have been tried from time to time. Almost every author has his favourite solution, to the use of which he has become accustomed. The reader may be referred to a detailed bibliography of the history of injection treatment by Junghanns.

It may be asked whether any treatment can be aimed at the real cause of the disease. To this the answer must be that neither surgery nor injection methods come strictly under this heading. Both (at any rate when the injection is used merely as a substitute for the knife or cautery) must be regarded as symptomatic remedies. They are designed to relieve particular symptoms of the rectal

varicose syndrome, the underlying cause remaining unaffected, just as the cause of dental caries is unaffected by filling a tooth. Surgical therapy cannot possibly affect the factors which actually produce the rectal varices. The so-called 'radical' operation is in reality even less radical than injection treatment. Indeed, this operation, which often consists largely in removing harmless skin-tags, shows how little understood is the true nature of the complaint. The conception of 'external' and 'internal', or 'subcutaneous' and 'submucous' piles, and the treatment that goes with this conception, can never be in the true sense aetiological. A treatment, if it is to strike at the cause of hæmorrhoids, must be directed against the hepatic and portal congestion and stasis.

It is not within the author's province to suggest methods of attaining this object. It is necessary to draw, somewhere, a line between what is the work of the surgeon and what properly belongs to internal medicine. The physician must play his part by seeking to devise in the light of accumulating knowledge effective methods of prophylaxis. In the author's series of 3100 cases of piles treated in the last four to five years almost 1 in 5 had already been treated by operation from one to five times. The injection method, as we shall show, has a much lower recurrence rate (and therefore should be considered the more 'radical' procedure of the two), yet recurrences do undoubtedly occur, and the reduction of this risk of recurrence is to be attained by a suitable regulation of the patient's diet and mode of life. The author's task in the present work must be limited to describing the technique developed by him for injection therapy and to discussing the indications and contra indications for operative interference. This last subject has led to much disagreement between the adherents of the two (fundamentally similar) methods. The differences of opinion are largely due to imperfect knowledge of the subject, and our aim is to clarify this situation. It must be added that in the writer's view the treatment of piles must remain in the hands of the surgeon.

It is a mistake to believe that the introduction of injection therapy has supplanted operation, for the latter is still in certain cases the method of choice. The injection is simply an additional weapon, and must be ranged along with the side-window proctoscope and the accurate measurement of injection solutions, both essential to good results, and together enabling us to deal more successfully with the disease. In the interests of his patient, the surgeon should be willing,

and able, to use all available methods. In the treatment of a single case, it may be necessary to make use of several methods as the clinical picture alters.

An example will serve to illustrate this point. Suppose that a patient is seen who gives a story of the appearance, with great pain, of a little lump at the anus. Inspection verifies the presence of a thrombosed nodule. He is in severe pain, cannot sit down comfortably, and for several nights has had no proper sleep. His condition demands immediate relief, and this can best be secured by incising the nodule and evacuating the clot. An injection of 5 c.c. of 1 per cent procaine deep to the nodule produces anæsthesia with instant

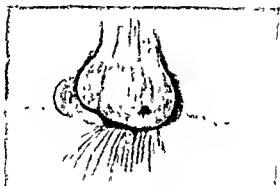


Fig 6 Thrombosed nodule

relief of pain, and an incision $\frac{1}{2}$ to 1 cm. long suffices to release the clot (*Figs 6, 7*). With the idea of avoiding infection, some surgeons prefer the diathermy needle for this incision. Though this may be better in theory, in practice the knife gives a better result, for a diathermy incision takes much longer to heal. The knife incision will heal without any reaction in three to four days, whereas if diathermy is used, eight to fourteen days may be required. Although the patient is now without pain and symptom-free he must not be regarded as cured. Actually, we have only relieved one symptom, namely an external thrombosis. A few days after the clot has been evacuated, treatment should be commenced for the internal varices. If this is not done, the patient may return sooner or later with another

thrombosis, or with 'prolapsing piles' His story will then be that the prolapse followed directly upon the last dose of a laxative, which he may have been accustomed to take for years Alternatively, he might return with an anal fissure Yet another possibility, if the underlying varices are still untreated, is that he may come with a prolapsed and strangulated 'polyp'—really the remains of a thrombosed internal pile, which again will obviously need a different type of treatment It is clear that a disease with such complex and varied manifestations can never be treated by a stereotyped routine, the methods must be adapted to the needs of the individual case In the treatment of fistulæ especially, it will presently be shown that



Fig 7—Showing the automatic evacuation of the thrombi when the noduli are incised

scalpel, diathermy, injection-syringe and proctoscope, sharp spoon, and astringent pastes and fluids all have their part to play The doctor who neglects to master the use of the whole armamentarium is certain to get into difficulties, and will eventually have to call in expert assistance

Injection therapy is therefore not to be thought of as a rival to surgery, nor must it be used indiscriminately in all cases, the aim and purpose of this book is to point out the proper indications for using the scalpel, diathermy, and injection

Turning now to the method of injection developed by the author, it must be pointed out that the term 'obliterative' cannot strictly be applied to it The adjective certainly described aptly

enough the results of applying to noduli destructive caustics such as fuming nitric acid, or injection of zinc chloride or ferric chloride. It might even be applied to Boas's method of injecting alcohol into piles drawn outside the anus by suction, because this leads to necrosis and cicatrization. These methods do undoubtedly produce some local thrombosis and shrinkage of the individual hæmorrhoids, but though they have long been familiar we have now abandoned them because they appear to us theoretically unsound, and moreover cannot always be carried out upon out-patients. Another objection is that the Boas method is only applicable to patients with a weakened sphincter, and only the tissues which can be made to prolapse can be treated. It is of no value in the early stages, where the sphincter is hypertonic or hypertrophied. Our method, which is essentially a proctoscopic one, is best described by the German word 'Abdrosselung', which means literally that the veins are 'strangled' or compressed by the fibrous tissue formed in the submucosa and can no longer dilate to allow a reflux of portal blood.

A series of injections placed by the author's technique submucosally round the lumen of the bowel at a high level may restore to normal within a few days even a severe mucosal prolapse. This in itself is sufficient to show that thrombosis plays no part nor is there any destruction of tissue for there are no visible changes at the site of the injections. The effect is produced by a chemically induced contraction of the submucous connective tissue with consequent compression of the contained veins. These changes tend to hinder or prevent any backflow from the portal to the caval system. Our experiences with this 'vein compression' method have led us to forsake the old 'obliterative' technique although even the latter could show temporary results quite as good as those of operation. In my view the systematic compression of the varicose veins by the submucous injection of quinine is the method of choice most nearly restoring true physiological function and leaving no scarring.

In England and America it is already realized that as the author holds this treatment should be in the hands of men specially trained in the pathology and therapy of rectal diseases. Thrombosed external noduli may, for instance, be associated with a carcinoma higher in the rectum, and failure to diagnose the latter is a serious matter. Certain firms of manufacturing chemists have issued misleading booklets of instructions for injection therapy, and have thus brought

the method quite unjustifiably into disrepute in some quarters in Austria and Germany

A few authors have recommended that 'external piles' should be injected with glucose solution and 'internal piles' with quinine. This suggestion is ill-founded, and based on insufficient clinical observation. Quinine must, it is true, only be injected submucosally, but it should not be put into the piles themselves. It should be placed circularly round the bowel above the hæmorrhoidal swellings. This may be called the site of election. If this is done accurately, in most cases treatment of external swellings will be unnecessary, for the latter are only of importance when they can be distended with portal blood by a reflux from above. If they are injected with glucose, tender œdematous swellings may persist for months. The author has seen a number of cases in which necrosis of these harmless 'external piles' had been produced in this way, and himself had a series of cases with local damage before learning the correct procedure and dosage. It is proverbial that each beginner must make his own mistakes and learn from them. As regards surgery, Langenbeck's operation is often undertaken for removal of even the most harmless skin tags, along with the lowest portions of the mucosa. The really important varicose veins lying 6 to 8 cm. above the anus, tortuous valveless vessels liable to become distended with portal blood, are quite out of reach of the operation, even though this may be repeated several times. Such treatment may result in stenosis, or in weakening of the sphincter with prolapse of the mucosa, and these failures show that this type of operation cannot truly be described as radical.

Treatment by injection around the higher varices is to be preferred to operations on the lines of Langenbeck's or Whitehead's for the following reasons —

- 1 Because the risks of secondary hæmorrhage, and later of stenosis, incontinence, or prolapse, are thus obviated
- 2 Because the higher varices are unapproachable by operation
- 3 Because operation requires hospitalization with consequent expense and loss of time
- 4 Because of the painfulness of the after-treatment (e.g., rectal tube and aperients)
- 5 In some recurrent cases because operation may be difficult or impossible

Even for advanced mucosal prolapse operative intervention is

now by no means always necessary, for such cases often yield to treatment by injections carefully placed 6 to 8 cm above the anus. Such treatment has obvious advantages over any operative procedure so far devised.

The chief arguments which have been advanced in favour of operative treatment for piles may be summarized as follows —

1 That operation permits the immediate removal of infected noduli and crypts under the anal skin without risk of subsequent thrombosis

2 That no sloughs are produced

3 That benefit is immediate

4 That cure is permanent, while the benefit of injection treatment tends to be temporary only

These points may be considered critically in order —

1 Infected piles (that is, those which have prolapsed and thrombosed and become secondarily necrotic) and external noduli are not in any case suitable for injection. They should be removed with the diathermy.

2 Injections also produce no sloughing if properly placed, of suitable composition, and accurately measured.

3 It is not entirely true that benefit after operation is immediate, there may be symptoms for some weeks afterwards, and certain manifestations of the varicose syndrome, such as constipation, may not be cured at all by operation.

4 Recurrence, as evidenced by our own cases, is certainly much commoner after operation than after injections.

Norman S. Kilbourne, of Los Angeles, has collected records of 62,910 cases by means of a questionnaire circulated to 293 leading proctologists in America, Great Britain, France, and Germany. The author describes the organization of the questionnaire as follows —

' On the suggestion of Curtice Rosser, the American proctologists were taken from a list of members of the American Proctological Society, together with men who, though not members, are included in its list of approved proctologists. Through the kindness of Mr. St. George B. D. Gray, of Hove, England, a list of members of the Subsection on Proctology of the Royal Society of Medicine was used. Six French and three German proctologists were included.

Of the many replies received, 57 replies gave definite information. Of these nearly all, i.e., 49, came from the American list of proctologists. It was agreed that the names of the individual contributors should not be mentioned in the final report so that each clinician might feel free to write with utter frankness about his own bad results.

Nearly all of the correspondents, 49 out of the 57, gave statistics on their personal use of both operative and injection methods

The total number of cases reported as treated by the two methods is by operation, 36,648, by injection, 26,262

In reply to the question, "What is your method of choice?" answers were *now use operation exclusively*, 11, *prefer operation but also use injections*, 12, *use both methods very extensively or choice depends upon the type of case*, 18, *injection is the method of choice*, 16—Total, 57

Operative Methods—The methods of operative removal were ligation and excision, 25,198 cases, clamp and suture, 2570 cases, cautery, 5779 cases, high frequency, 101 cases

Injection Solutions—The solutions used for injection according to the number of proctologists using each are quinine urea hydrochloride (usually 5 per cent) 23, phenol in oil (usually 5 per cent), 11, both quinine urea HCl and at other times phenol in oil, 8, phenol in glycerin 3, alcohol (70 per cent), 3, alcohol, ergot, and phenol, 1, double chlorhydrolactate of quinine and urea in glycerin 5 per cent, 1

Mortalities—In the 33,648 cases treated by ordinary operative methods there were no more than 11 mortalities

In the 26,262 cases treated by injection there were no mortalities that could in any way be attributed to the injection treatments

Sloughs following Injection—One proctologist, whose experience in injection methods was limited to 57 cases, tried various solutions and reports sloughs following injection in every one of the cases. In 26,205 patients treated by 42 other men there were 228 sloughs of importance. This makes a total of 285 more or less serious sloughs in 26,262 cases or an incidence of about 1.09 per cent. How serious these sloughs were may be judged from the results as to hæmorrhage and stricture which follow.

Hæmorrhage—After operations on 31,950 patients there was serious post operative hæmorrhage in 183 patients, or 0.573 per cent.

After injections used on 26,183 patients there was serious hæmorrhage in 73 patients. Of these, 28 were in the practice of the doctor who had sloughs in every one of his 57 cases. Including these we have in the whole series a percentage of serious hæmorrhage after injection of 0.279 per cent.

Stricture—After ordinary operations stricture followed in 68 cases out of 30,925, or 0.22 per cent.

Stricture after injection methods occurred in 6 cases out of 26,183, or 0.02 per cent. Five of the six post injection strictures occurred in the practice of the proctologist who reported sloughs in every case injected. The other case of stricture followed the use of quinine and urea hydrochloride.

The statistical treatment of the recurrence-rate is not easy. A return of symptoms after operation is often not strictly a recurrence at all, but the result of incomplete operation. The same high lying varices still persist, unreached by the surgeon.

The author's personal series of some 3100 cases is of considerable interest. He may with some reason claim unbiased judgement, having practised operative treatment for twelve years at four surgical clinics before adopting injection methods, and afterwards having had the opportunity to observe both his own results and those of others. Thus ample first-hand evidence of both methods has been obtained. Moreover, whereas the author's 300 operation cases were spread over twelve years, the injection cases are all recent and fresh in mind, so that the difficulties and complications in the latter group tend to bulk more largely than in the former. In spite of this, the author's impression is that there were no more mishaps with the 3100 injections than with the 300 operations. The patients themselves (apart from any statistical or medical opinion) undoubtedly pronounce in favour of the vein compression method.

CHAPTER V

TECHNIQUE OF TREATMENT

EXTERNAL skin tags must never be injected. If they give trouble on account of their size, or if they interfere with cleansing, or indeed if the patient wishes it, they may easily be removed in the out-patient department or consulting-room, one or two at a time, by diathermy. The technique is as follows. The tag is anesthetized with 2 to 3 c.c. of 1 per cent procaine, with the patient lying on his right side, the tag is now grasped in forceps and its base crushed with a clamp. The clamp is then removed, and the tag cut off with the diathermy needle at the groove thus made. The operation is aseptic and bloodless, and makes ligatures or suture unnecessary. Not more than two tags should be dealt with at each sitting. Many doctors having no personal experience of the treatment are led by the instructions published by manufacturing chemists to inject all redundant tissue at the anus. As a consequence, we have seen a number of patients with commencing necrosis of the tags. Even at this stage, the moist and painful tag may be removed in the manner described above.

Thrombosed external varices should generally be incised. Local anesthesia is obtained as above, and as a rule the thrombus delivers itself through the little incision. Sometimes thrombosis may recur after one or two days at the original site or close by. If this occurs in the same varix, the clot may be expressed through the same incision with a small swab, without further anesthesia. The author had one patient (among many hundreds) who had a recurrence at the same site every day for three days in succession. After operation there is no need for a rectal tube or aperients nor need any special instructions be given. The next stool is usually quite painless. As a rule the thrombus which may be moderate or large in diameter, is single, but occasionally there may be several small thrombi which cannot be reached through the same incision. In such cases five or six tiny incisions, 2 to 5 mm. long, may be required (*Fig. 8*). In cases where the thrombosis has existed longer than eight days the treatment is a little less simple, for commencing organization will have

caused the clot to adhere to the varix wall. It will require forcible removal with a pair of forceps, and the whole varicosity may have to be torn away from the afferent and efferent vessels, or perhaps the clot may be snipped away with a part of the vein wall. A gauze pad is sufficient dressing. To avoid waste of time, the proctoscopic examination may follow immediately upon this operation, it can now be done without discomfort because anæsthesia is already present. *Where there are external thromboses there will always be internal varices,*



Fig 8 Thrombosed external varices. The thrombi have already been removed

and there are no exceptions to this rule. Spontaneous thrombosis, the aetiology of which will be discussed more fully in a later chapter is not merely a casual accompaniment of internal varices but is the direct result of a reflux of blood from these higher varices. Hence the treatment of the latter should follow as soon as possible upon the cure of the external thrombosis.

The internal treatment is best carried out by means of a side-window proctoscope such as Blond's (Fig 9). The experienced proctologist can immediately distinguish normal from abnormal

mucosa The pathological appearance of the mucosa in 'proctitis', with its clearly marked vessels, is reminiscent of the appearance of gastritis, indeed it seems to us quite possible that this latter condition may also be a result of portal congestion The injection is given through the side-window of the proctoscope, the finer the needle used, the less the patient will notice it It is important that the injection be placed at the optimum site, and this question must now be discussed In our opinion, *the contention that the aim of the injection should be to produce thrombosis of the varices is erroneous* Success does not depend on clotting, indeed it is scarcely possible to induce clotting in the higher veins, which belong to the portal



Fig. 9. Injection through the proctoscope

system for these vessels are very resistant to irritant substances It would appear that the intima of the portal radicles forms certain antibodies to combat noxious substances absorbed from the intestinal mucosa Moreover, the small amount of solution injected (2 to 3 minims) would become so rapidly diluted in the vein that its effect would be negligible Most experienced and observant writers agree that the injection should not be intravascular It should be placed deliberately in the submucosa Thus Bensaude and Oury, for example, infiltrate the entire submucosal layer with a 5 per cent quinine urea solution The present author has tried out a large number of preparations which he has systematically tested and

evaluated, his final preference being for a quinine solution with the following formula —

| | | | |
|---|-----------------------------------|-----------------|-------|
| R | Quinine bihydrochloride | g | 30 |
| | | (now reduced to | 20 g) |
| | Urethane | g | 14 |
| | Procaine | g | 2 |
| | Tincture of catechu (20 per cent) | cc | 15 |
| | Water to | cc | 100 |

Since catechu has a pronounced astringent effect, the compression of the larger veins is brought about more rapidly than with a simple quinine solution of like concentration

The injections are placed round the bowel in a clockwise sequence, under the mucosa at a level above the hæmorrhoids. Each injection consists of not more than 1 to 2 minims of solution, and not more than 8 minims should be used at one sitting. The higher the injection above the anus, the less the patient will feel the prick of the needle. The ring of injections which may take four to five sittings to complete, should be situated about 6 to 8 cm above the anus. In cases of proctitis and pruritus ani, however, the treatment is different and will be described in a later chapter.

The patient should not be treated more than once in the first week. Some patients have so marked a reaction after the first treatment that even longer must be allowed for it to subside. As a rule, however, the reaction becomes apparent only after the third treatment. It is advisable to use not more than 3 to 6 drops at the first treatment, in order to gauge the patient's susceptibility to quinine.

Cases with very large internal hæmorrhoids may prove very resistant to the treatment, and in these considerably larger doses are necessary. One of our patients, a man of 70 required more than twenty treatments before the almost fantastically large varices were controlled. Generally, after the third treatment the bleeding, pain or prolapse disappear and do not recur. The cure is usually complete in five to seven treatments. On the day after the first treatment some patients complain of rectal discomfort, some complain of slight pain which lasts for a day, others again experience no unpleasant sensation whatsoever. After the third treatment a number of the patients complain of a sensation as of a foreign body in the rectum, which persists for eight or ten days and is then succeeded by a feeling of relief and well being. By far the greater proportion will state that they feel as though they had a new lease of life. If a patient complains of tenesmus, or the sensation of a foreign body in the

rectum, treatment should be discontinued, and only resumed (should more be needed) when the symptoms disappear. After an interval of six weeks or so, proctoscopic examination should be made to determine whether a further series of treatments is advisable. Recurrences are generally the result of insufficient treatment. Many patients, especially doctors, cease treatment the moment their symptoms are relieved and only return when further bleeding, or a thrombosis or prolapse, demonstrates that treatment was never completed.

An unpleasant complication is the quinine rash, which involves the whole trunk and may be accompanied by a high fever. A few drops of quinine may be sufficient to produce this eruption in susceptible individuals. In spite of the alarming symptoms, especially the high temperature, recovery is rapid and will be complete in eight to ten days providing treatment with quinine ceases. For such cases a concentrated glucose solution (33-66 per cent) may be used. In 1935, after some 2000 cases had been treated without serious complication, there occurred in rapid succession two fatalities, in the following circumstances:

The first case, a woman of 54, came to my private clinic on June 18 1935, complaining of an external thrombosed pile. The nodule was anesthetized, on June 21 she came again and the nodule was collapsed, and not tender on pressure. She then received an injection of quinine urethane solution submucosally in several places, *a total of 0.5 c.c. being used*. On June 25 she returned for another treatment, 0.5 c.c. again being given. She was asked to return in eight days for the third treatment, but instead came in three, on June 28, as she wished to leave town and was again injected with 0.5 c.c. of solution. Soon after leaving the consulting room she came back complaining of bleeding. She had certainly bled a little, but as proctoscopy showed that the bleeding had ceased she was allowed to go home. Two days later, at 8 p.m., she began to have severe pain. A doctor was called and she was given morphia and suppositories. The writer was next called and found the patient under morphia.

Examination revealed a small painful ulcer no larger than a fissural ulcer on the posterior wall of the rectum. He advised continuation of the suppositories and promised to call the following day. That night yet another doctor was called in, however, and he sent the patient into hospital immediately with a diagnosis of a perforated appendix. The patient entered hospital almost pulseless and in spite of a blood transfusion died the same day, July 1.

Autopsy Findings—The diaphragm was at the level of the 4th right rib, and the 5th left rib. The abdomen contained a small quantity of turbid reddish fluid. The serosal covering of the gut was lustreless in many places, and the gut somewhat distended. A quantity of free fluid

was found in the pouch of Douglas there was no gross lesion of the peritoneum of the pouch of Douglas, and the inferior mesenteric and pelvic veins were healthy

In the rectum about 3 cm above the anus on the left side, there was a defect in the mucosa twice as large as a lentil bean. It penetrated at one part to the inner muscular layer, and had a ragged necrotic margin. A probe could be passed to a depth of about one centimetre. The surrounding mucosa was œdematous, purplish, and in places deficient, just above the ulcer it had a pale grey appearance. The submucous veins were somewhat enlarged, and not thrombosed. The perirectal cellular tissue, especially on the left, was markedly œdematous, discoloured, and infiltrated with pus. The changes extended extraperitoneally to the upper limit of Douglas's pouch. Other than this, the gut was healthy. There were two small uterine myomata. Both lungs were free œdematous, and autolytic, the lower lobes were congested posteriorly. The heart was enlarged to about one and a half times the normal. The myocardium was very flabby, pale, and friable. There was one calcified lymph-node the size of a cherry at the hilum of the right lung. The thyroid showed a few small colloid nodules. The bile-ducts were healthy the liver was autolytic, with yellowish mottling. The spleen was rather soft, and double normal size. The suprarenals contained lipid. The kidneys showed cloudy swelling and autolysis. A smear preparation of the peritoneal exudate showed mixed organisms, streptococci predominating. A smear of the cellulitic area contained chiefly streptococci, with some Gram positive bacilli.

Pathological Diagnosis—Early peritonitis. Left lateral perirectal abscess, due to a rectal ulcer. Internal hæmorrhoids. Pulmonary œdema and emphysema. Cause of death septicæmia (Dr Balzar)

The second case, also a woman of 54, was first seen on June 19, 1935. A diagnosis of hæmorrhoids and pruritus was made. She received her first treatment on June 25th with 0.5 cc of solution, and the second three days later. The following day the writer was called to the patient's home and found her in bed with intense rectal pain. The abdomen was distended and there was diarrhoea. She was exhausted from lack of sleep. Digital examination of the rectum revealed a definite ulcer, and proctoscopy showed that this was due to a localized necrosis of the mucosa. Two days later the patient was admitted to hospital with all the appearances of a rapidly spreading pelvic cellulitis.

The following are extracts from the patient's notes at the hospital (Denk) —

A. B., female, 54. July 5 to 19, 1935

Local Condition—Proctoscopy shows thrombosed hæmorrhoids 7 cm above the anus, and on the left rectal wall a necrotic area the size of a lentil bean. The superficial slough can be removed by wiping with a swab. Digitally, the ulcer admits the tip of the finger.

Abdomen—Belly wall soft and flaccid. Spleen and liver not palpable. Slight tenderness on pressure over the whole lower abdomen, and marked tenderness in the region of the true pelvis.

General Condition—A large, strong, well nourished woman Colour slightly yellow Tongue rather dry

Urine—Albumin present Deposit contains a few leucocytes No sugar or urobilin

Subsequent Progress—

July 6—Short wave diathermy treatment Temperature settling Rectal bleeding, arrested with 'stryphon' suppositories No appetite Retention of urine, catheterization

July 9—Further considerable rectal bleeding, suppository

July 10—Blood count Red blood count, 4,100,000 Hb (Sahli), 80 per cent Temperature again higher, 102.6° F Retention continues

July 11—Blood transfusion of 500 c.c. (Group II) Digital rectal examination shows cavity the size of a mandarin orange deep to the mucosa of the anterior rectal wall, projecting into the vagina, which is not perforated It extends as high as the pouch of Douglas

July 12—Operation under nitrous oxide anaesthesia Incision as for Kraske's operation The coccyx was excised and by parting the soft tissues the rectum and vagina were exposed An abscess cavity was disclosed to the right of the rectum about 8 cm above the anus, containing foul-smelling pus and blood and shreds of necrotic connective tissue The abscess had already perforated the visceral layer of fascia The state of affairs was now seen to be as follows On the anterior and right walls of the rectum was an opening the size of a halfpenny, with irregular margins This led into a cavity about as large as a hen's egg full of necrotic material and blood, with poorly demarcated boundaries It was situated in the pelvic cellular tissue and extended as high as the pouch of Douglas The cavity was thoroughly cleansed with hydrogen peroxide and normal saline, packed with iodoform gauze, and the wound partially closed with sutures Bleeding during the operation was insignificant

July 13—Patient very drowsy

July 14—Gauze plugging changed

July 15—Patient is apathetic but accepts fluids Urine albumin strongly positive

July 16—Sudden onset of bilateral parotitis in the morning Temperature rising to 101° during the afternoon Parotitis becoming worse

July 17—Parotitis continues Patient very drowsy

July 18—Bladder wash-out Incision of parotid gland

July 19—Patient died at 9.30 a.m.

Autopsy revealed extensive foul pelvic cellulitis

That the necrosis of the mucous membrane may have led to the rapidly spreading infection of the pararectal tissues is unfortunately possible The complication is of course extremely rare, having occurred only twice in our series These two cases, nevertheless, constitute at first sight a serious objection to the method It is possible that the risk of necrosis may be eliminated by altering the

solution used, or its dosage (see Author's PREFACE) When such a seemingly safe treatment suddenly results in two fatalities one is certainly justified in feeling a doubt as to whether the old operative methods should not after all be preferred This doubt is, however, set at rest if the mortality-rates of the two methods are fairly compared Previous statistics seem to us to have been unfair for the following reasons —

1 The cases treated are by no means similar One has to think of the probable mortality-rate in an operative series including as many prolapsed cases over 60 years old as we are accustomed to treat by injection, for in such cases the results of injection treatment are eminently satisfactory

2 Operation as usually practised is incomplete, in that it does not remove the higher-lying varices If these varices were attacked (either by excision or cauterization) the operation would last longer and the mortality would necessarily be higher In our series we have treated several medical colleagues who had been previously operated upon from two to four times without relief of pain or cessation of bleeding In fact, there were cases with high varices actually suffering from secondary anæmia as a result of continued hæmorrhoidal bleeding occurring since their operation Yet these cases responded very quickly to injection treatment, with complete cessation of bleeding Thus it is evident that the indications for injection treatment are considerably wider than for operation Deaths resulting from secondary anæmia after hæmorrhoidectomy should in fairness be added to the operative mortality

3 Where operation is contra-indicated, as for example in patients with heart, liver, or kidney diseases, or in advanced old age, successful relief of bleeding, prolapse, or pruritus can be given by injection

In spite of the great opposition of the medical profession, the injection method has therefore found great favour in France, England, and America since the last war, especially after Swinford Edwards had reported the results of his twenty-five years' experience in the *Practitioner* (1915) Since then the literature has contained numerous reports from proctologists who have used the method with success The following may be instanced Graeme Anderson, Terrel, Collier, F Martin, Hirschman (Detroit), Jackson, Alexander and Murphy, Montagu, Gordon-Watson, Lockhart-Mummery of London, Roux in Lausanne, Jose Jorde of Buenos Aires, Edmond Iscomel from

Lima, Meisen of Copenhagen, Berglund of Norway, and Hooton from India *

In Germany and Austria the credit of reintroducing the method must be given to Boas. He adapted the alcohol injections already used for varicose veins by Monteggis in 1913. In these countries, however, the method was slow to gain adherents. Kirschen was the first to adopt Boas's method in Vienna. The French, and in particular Bensaude and Oury, recognized the shortcomings of Boas's technique and did not make use of it.

It is notable that almost all publications on the subject of injection therapy are entirely without any attempt to understand and explain the underlying scientific principles. The author, in a series of papers, has tried to make clear the theoretical basis for the 'vein-compression' method, and has shown that though previously limited to the treatment of hæmorrhoids, it could well be extended to the treatment of all the manifestations of the rectal varicose syndrome. This widening of the indications, and the success obtained in treating *pruritus*, *fissure*, *fistula*, and *habitual constipation* have led to the formation of certain new theoretical concepts with which this book is concerned. The experience gained by the author in the past five years gives good grounds for hoping that the scope of the therapy may be yet further increased. Just before the completion of the original edition of the book in 1935 the two fatal cases of pelvic cellulitis occurred, and these at first seemed a severe setback to the method. Up to that time, the author had seen no serious complications, and the occasional reports in the French literature of quinine intolerance were considered by him to be the unfortunate results of excessive dosage or faulty technique. But in the light of these two tragic cases the reports have come to possess a much greater significance.

A Achildi, for instance, reported the following case of intolerance to quinine bhydrochloride with urea —

A woman, aged 25, was injected with a quinine-urea solution (Rinnrea) into a congested hæmorrhoid. The amount used was about 0.015 g. The injection was immediately followed by a feeling of tension about the mouth and some swelling of the lips. This woman—she had been accustomed to take quinine since the age of 12 for malaria—noticed a taste of quinine. Her face became erysipelatoid in appearance, the eyelids and lips œdematous, the conjunctivæ injected, and the pupils dilated. There was disturbance of vision, and urticaria over the whole body. There was icterus, and

* All cited from Bensaude and Oury, *Les Hémorrhoides et Leur Traitement*

abdominal pain with hyperperistalsis and a pulse so rapid as to be uncountable

Recovery, fortunately, followed in this case after two days, but an earlier case reported by the same author had proved fatal. In view of such cases, we must reassess the apparently trivial symptoms occasionally noted by patients the day after the injection. Since only a minute percentage of the cases treated have these untoward symptoms, we believe that they must indicate extreme susceptibility to quinine. These patients have complained of the sensation of a foreign body in the rectum, of great increase in the size of the hæmorrhoids, and all treatment has had to be stopped for eight or ten days. Some have developed a rash after only a few drops of quinine, with fever rising perhaps to 103° or 104° F., or sometimes disturbance of micturition lasting a few days. There may be extreme fatigue, and pains radiating to the lower limbs. Several cases have developed local necrosis of the rectal mucosa. During the last two years we have tried out many preparations of quinine, our experience coincides on the whole with that of most writers that the concentration of the solution is by no means the only factor. Even with a 5 per cent solution necrosis may occasionally occur. Bensaude refers to his seven years' experience in the treatment of piles with sclerosing solutions as follows —

To 325 patients treated (226 men and 99 women), 2753 injections in all were given. The author reports 45 per cent of cures, 47 per cent improved, and only 3 per cent of complete failures. The technique consists of a submucosal (that is, interstitial, not intravenous) injection of a 5 per cent solution of the double chloride of quinine and urea. The injections were given by direct vision with the aid of Bensaude's "anuscopé" above the piles. At each sitting, only one injection of 3 to 5 c.c. was given. This was repeated at intervals of eight days until the whole circumference of the bowel had been treated and sclerosed. Bensaude emphasizes that the method is only applicable to *internal* hæmorrhoids. It is suitable also for cases of prolapse providing the sphincter is active. If the injection is given too superficially it may readily lead to a localized necrosis of the mucosa. The mistake can usually be detected during the injection for the mucosa swells up into a pale bleb full of fluid. In this case an effort should be made to express at least a portion of the fluid. Equally important, in the author's opinion, is that care should be taken not to place the injection within a vein.

Thus necrosis may be encountered even with a 5 per cent solution. It must next be considered whether such areas of necrosis are really important as portals of entry for spreading cellulitic

infection. The two deaths in our series seem to suggest this possibility, but on the other hand the following facts must be considered —

1 Mucosal necrosis may occur spontaneously in a case of piles if these prolapse, and if replacement is delayed, yet with very rare exceptions this does not lead to pelvic cellulitis.

2 Langenbeck's cautery operation produces deliberate necrosis, yet even though this does not extend above the sphincters fatal infection may occasionally follow.

3 Since the injection method has widened the scope of treatment to include high lying varices and cases of advanced prolapse, it is reasonable to object that necrosis high in the rectum may be more dangerous than a similar occurrence low down. Yet on the other hand it must be asked whether the mortality would not be much greater still if these advanced cases were submitted to operation. Any increase in the scope or severity of the operation must inevitably lead to correspondingly increased risks.

These considerations, however, have no direct bearing on the question of quinine susceptibility as a possible source of danger which it would be impossible to eliminate entirely. Fatal cases of perirectal cellulitis are known to occur quite apart from operation or injection. Such infections have a latent period of three to five days before the diagnosis can be made with certainty. As already stated, our observations indicate that the infection arises in a thrombosed and secondarily infected varix. Now in the large town of to day every patient with rectal troubles soon learns from his neighbours that relief may be obtained without operation, so that there is a tendency for sufferers first to come for treatment when their previously mild symptoms become suddenly worse just such a change as may be brought about by a thrombosis and commencing infection in a high varix. Thus the patient may come to the doctor with a commencing though not obvious perirectal infection, and be given an injection. Obviously this injection cannot be expected to benefit the already established infection. That such cases do occur is proved by a recent example, a patient who came to me shortly after the two fatalities already described.

A man of 56 was sent to me on July 13 1935 complaining of rectal pain. The proctoscope showed well marked internal varices and save for slight pain on withdrawing the instrument nothing unusual was observed. No sign of a fissure could be found, but on repeating the digital

examination, a definite tender longitudinal ridge was discovered on the posterior wall, resembling an ordinary thrombosed vein. Because of the two recent fatalities I refrained from any local treatment, and ordered suppositories. Two days later, on July 15, there was an obvious perianal inflammation, which was treated surgically. I learned from the patient's doctor that he was able to resume work the next day. For a week he lived his normal life, and as he had no symptoms he did not return to me as instructed. On Sunday, July 21, he took part in a picnic, and on his return noticed rectal pain. Next day he went to work but returned home at midday with shivering attacks. His doctor sent him immediately into hospital, and he died on the same day of septicæmia.

Had this patient received an injection, the disaster would have been laid at the door of the treatment. Undoubtedly such cases must occur from time to time without the true sequence of events being recognized. This diffuse, septic type of periproctitis is almost always fatal in from two to ten days. Moreover, it is not quite as rare as is generally thought. In some circumstances the true state of affairs must remain obscure, and unwarranted blame be laid on the injection method.

In spite of all these considerations, it is probable that true quinine intolerance does exist, though it is extremely rare, and that it occasionally leads to fatal accidents. Where the treatment is less well-established than in France and England and America, there is a risk that its opponents may make undue capital out of these mishaps. We therefore advance the following points in defence of the therapy —

1 Every new method has its failings and its accidents, even the injection of varicose veins has given rise to tragic fatalities, yet it is held in esteem by the whole profession.

2 As we shall show, the success of the method in cases of otherwise intractable pruritus and eczema alone makes it indispensable. The 175 cases of fistula we have succeeded in curing would alone suffice to justify its use. Some of these last cases had been treated by surgery three, four, even five times without success, though the operations had brought the patient to death's door.

3 The relief of habitual constipation in many hundreds of cases is a strong argument for the treatment.

What is needed is an exhaustive and critical analysis of all the complications and fatalities reported in the whole of the literature. Their causes would then be recognized and if possible eliminated. In consequence of the two fatal cases in our series, we have adopted the following modifications

1 We have reduced the strength of the quinine solution from 30 to 20 per cent (*see* Formula on p 28)

2 In the first treatment, not more than 5 minims is used, to guard against possible quinine idiosyncrasy. Formerly, as much as 20 minims was sometimes given

3 An interval of five to eight days is allowed, to make certain that there is present no incipient perirectal infection (*see* Preface)

4 Not more than 9 minims is now injected twice each week. This amount contains 0.05 g of quinine, distributed in separate foci of 2-3 minims (0.01 g) each

5 In cases with quinine idiosyncrasy, or where the hæmorrhoids are very large, a glucose solution is used in doses of a few drops. So far no complications have been observed with this solution

That the toxicity of the quinine solution hitherto used by us must be extremely slight is shown by the fact that in 200 fistula cases 1 to 3 c.c. has been given at a sitting without a single mishap. It is our belief indeed that the quinine only leads to necrosis if injected into an already thrombosed vein. If the clot is already infected, necrosis and sepsis follow rapidly. Otherwise we believe the solution in this dosage is perfectly harmless, whether injected submucosally or subcutaneously (*see* section on treatment of PRURITUS, p 73). The pathological quinine-susceptible case must be excepted from this generalization

CHAPTER 11

ANAL FISSURE

THIS condition was known to the ancients. In later times, Ambroise Pare in the sixteenth century made important observations upon the disease. In 1848-9 Boyer, and in 1868 Rodenhammer, published their therapeutic experiences. Gant recognized quite clearly that this linear crack was not really a tear of the anal mucosa. If the anal folds are straightened out, it can readily be seen that the 'crack' is in reality circular in outline. Gant therefore coined the term "painful anal ulcer."

By anal fissure, then, is understood a painful ulcer at the mucocutaneous junction. The pain comes on in attacks, accompanied by spasm of the sphincter, and sometimes of the whole of the neighbouring musculature. The levator ani may be involved, in which case the pain will spread to the sacral and bladder regions. The ulcers are generally single, two being rarely seen simultaneously (thus in the author's series of 600 cases two lesions were observed in only 10), while in one case no less than three 'fissures' were present. The commonest situations are the posterior and anterior commissures but they are occasionally seen elsewhere. In depth, the lesion seldom or never extends more deeply than the vessels in the submucosa. In the majority of cases, the presence of the ulcer can be determined at the first glance. Its existence is betrayed by the so-called *sentinel pile*. This fold or prominence of the anal skin is simply the remnant of a previously thrombosed and ruptured or partially necrosed external nodule. This statement is an anticipation, for the subject of aetiology is dealt with later.

If the literature is searched for ideas about the causation of fissure, it will be found that faecoliths, or prolapse or injury by hard faeces or foreign matter (e.g. fruit seeds), are generally blamed. Haemorrhoids, too, are often given as a cause, yet even those writers like Bensaude and Oury who ascribe to haemorrhoids an important role state that internal piles are found in only 70 per cent of cases of fissure. These two authors have suggested that the fissure would be more accurately named '*varicose ulcer of the anus*'. This is fully

in accord with our own observations, but we must add that piles are to be found not in 70 per cent, but in 100 per cent of the cases. The fissure owes its existence solely to a thrombosed varix at the mucocutaneous junction. It is in fact merely a partial manifestation of the varicose syndrome. Whereas, for example, no less an authority than Gant claims that fissure plays an all-important role in the aetiology of constipation, it is our own view that the constipation itself is only another aspect of the varicose syndrome. Similarly other lesions which have been considered responsible for fissure, such as strictures, polypi, eczema, and anal pruritus, are but parts of the syndrome.

Quenu and Hartmann have also recorded that 70 to 80 per cent of fissure cases have in addition internal piles. The view that piles are a cause of fissure has been disputed on the ground that simple dilatation of the sphincter, though an efficient treatment for the fissure, can have no possible effect on the piles. This conclusion is illogical, for the cure of the fissure—that is, of one symptom—need not depend on the complete cure of the whole complaint. It is clear that the healing of the fissure, which is only one small part of the varicose syndrome, does not necessarily imply any improvement in the underlying varicose disease. Even bleeding itself is not constantly present in cases of piles, yet if bleeding is absent, or ceases, no one concludes that the hæmorrhoids are cured. During the course of the disease, the symptoms are prone to alter, and with the passage of time fresh symptoms in turn become predominant. Those previously latent may appear as others lessen, pain, bleeding, moistness, itching, mucous or purulent discharge, sphincter spasm, constipation, proctitis, erythema, eczema of the anus, scrotum, and vulva, thromboses, periproctitis, fissures, fistulae, prolapse, stricture, and many referred symptoms should all be grouped together as parts of the rectal varicose syndrome.

In the literature different interpretations have been placed on the symptoms which may accompany fissure. Boyer, for instance, thought that the sphincter spasm was the initial lesion, and the fissure its result, others have taken exactly the opposite view. The error lies in singling out one symptom of a complex and declaring that it is the cause of the others, whereas in fact the whole complex is dependent on some primary cause. Because many of these patients are constipated, some writers have come to believe that hard faeces produce fissures by tearing the anal mucosa, indeed that is the origin

of the term 'fissure' In this they have completely overlooked the fact that true tears neither produce the same attacks of pain nor exhibit the characteristic slow-healing qualities of so called fissure

Even the appellation 'anal ulcer' is not quite accurate In some cases the edges of the lesion may be seen to be pouting, inflamed and cedematous, and if a probe is used after anesthetizing the area, a track may be discovered leading from the floor of the 'ulcer' (Fig 10) Some older authors indeed wrote of 'fistula in ulcere' instead of anal ulcer In these patients the fissure usually produces a mucopurulent discharge, and the anal skin is constantly moist The edges

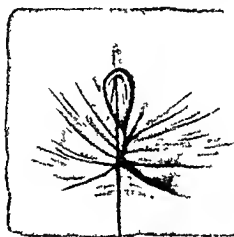


Fig 10 Anal fissure with fine fistula opening (ulcer fistula)

of the fissure are often undermined and the sentinel pile inflamed and tender on pressure

Acute and chronic stages of fissure may be distinguished In the chronic stage the pain is as a rule only moderately severe the chief sign of the presence of the partly healed fissure being the spasm (or, more accurately, the hypertonus) of the sphincter and In the acute stage, the pain may be so severe that the patient is quite unable to work or to take any pleasure in life Yet at times the pain may be completely absent True sphincter pain begins immediately after defæcation and lasts as a rule for one or two hours A typical story is that the pain radiates to the gluteal and sacral regions, and hitherto no explanation of this spread of symptoms

has been suggested. A probable explanation is as follows. Fissure does not exist without piles, it is always preceded by thrombosis in a varix, indeed the fissure is nothing but a localized necrosis of a small superficial thrombosed vein. The onset of thrombosis is accompanied by severe pain which is transmitted by the sympathetic plexus which follows the blood vessels. Since the fissure is a thrombosed and ulcerated nodule or varix, there are likely to be very small thrombosed veins running from the lesion to the larger veins which unite on the posterior wall of the rectum into big trunks. During defæcation, the levatores ani muscles will pull on and irritate these veins, thus causing the widespread pain.

Another important accompanying symptom is constipation. This, too, is a subsidiary manifestation. The conception hitherto has been that the patient avoids defæcation for fear of the pain. Against this view we may cite the following facts —

- 1 Defæcation is not entirely a voluntary procedure
- 2 The constipation does not diminish when the fissure is healed
- 3 The constipation may have existed before the fissure developed, indeed it is held by some to be the precipitating cause for the latter, by causing trauma from hard fæces

It will be seen how self-contradictory are such attempts at explanations, and how badly they withstand thoughtful criticism. Constipation itself is only a symptom, and disappears when the internal varices have been treated. Another symptom, almost always to be observed, is flatulence. Streaking of the stool with blood is also a common symptom, though fissures may sometimes produce severe bleeding. Gant has made an interesting observation which we have been able to confirm many times, and which supports the views we now put forward. He discovered that the amount of pus coming apparently from the base of a fissure had no relation at all to the size of the latter. If the anæsthetized ulcer is opened out, careful examination with a lens will disclose a little track opening in the floor of the ulcer or at its edge. Pressure from the anal canal *with a finger will express from the track a quantity of thick pus*. Gant wrote "In exceptional cases a fistula may have its opening in a fissure" (Fig 11).

Careful consideration will show that such a finding must be not exceptional, but the rule. If a fissure develops from a nodule—that is, a varix—which has thrombosed and afterwards undergone surface necrosis, we would expect to find traces of afferent and efferent

vessels. Generally these are so small that the lumen is not visible to the unaided eye. Nevertheless we have succeeded in demonstrating them in a number of cases and one case is recorded in the water colour by Hajek (*Fig 11*). Any careful observer may verify the fact for himself. It is only necessary to adopt a certain technique which we shall describe later. It is of interest that such a valuable observation made thirty years ago should have been completely forgotten probably because neither Gant nor later workers were able to interpret it correctly.

The nature of the pruritus in such cases has also been wrongly explained. It is said to be due to the



Fig 11 Double fistula of the rectum and anal canal

decomposition of the secretion which collects between the folds of the skin. The following facts however conflict with this theory. (1) There are discharging fissures which are unaccompanied by irritation. (2) Fistulae are often seen with much free secretion yet without the slightest itching. (3) The itching may persist undiminished after the fissure has healed. (4) Cases of pruritus are seen so severe as to lead to thoughts of suicide yet without discharge and without a fissure. The pruritus may subside with the healing of the fissure but not necessarily so. It is neither the cause nor the result of the fissure both are alike symptoms of the varicose disease always present in the rectum and anal canal. The fact has

been overlooked till now simply because surgeons have not taken the trouble needed to examine thoroughly the indrawn funnel shaped anus of these apprehensive patients.

That fissure cannot be a purely local lesion as many have thought is shown by the referred symptoms so commonly observed in these cases. Pain radiating to the male or female genitals (prostate testicle uterus or ovary) is often encountered. The sacral region the bladder or the lower limbs may also be the site of the pain. One of the most frequent accompaniments of fissure is dysuria which may even progress to complete retention. Lack of familiarity with this symptom of so called anal fissure may lead to quite unnecessary

operations. Many prostatectomies could be avoided if a proper examination were made with a proctoscope. Digital examination of the prostatic patient is insufficient, and may be misleading. *Many cases of so-called prostatic hypertrophy are really cases of acute congestion or œdema of the prostate due to impairment of its venous return.* The pain radiating to the bladder and genitals has never before been satisfactorily explained, but now its meaning becomes perfectly clear, and may be summed up in the following way. In more than 600 cases of fissure we have been able to watch the development of the ulcer, stage by stage, and to follow the process of healing in detail.

These ulcers are not confined to the mucocutaneous junction, but may be met with at any part of the rectal mucosa where spontaneous thromboses and subsequent ulceration of the clot through the vein wall occur. Sometimes it is possible to follow with the proctoscope the delivery of the clot through the opening which forms. First is seen a tiny breach in the mucosa overlying the varix, and in the wall of the vein itself, through which the red-brown thrombus may be glimpsed. Then as the necrosis proceeds, a part of the clot becomes clearly visible, and at last, generally at defæcation, it separates completely and disappears. In this process, therefore, the superficial wall of the varix and the mucosa over it are lost. The floor of the resultant ulcer (or fissure) is formed by the deep part of the vein, and its edges, usually, pouting and thickened, are composed of skin or mucosa with the adherent margins of the opening in the vein. Every thrombosed varix must have vessels connected with it, leading from the capillary bed, and these will also be thrombosed, usually right back to the corresponding capillary zone. Seeing these cases as we have done almost daily, and treating them by incising and drawing out the clot, we have had ample opportunity to study these points.

On the expressed thrombus it is often possible to make out quite clearly the position of the afferent and efferent venules. Now the branches of the inferior hæmorrhoidal plexus communicate with the pudendal plexus, which receives the venous blood from the genital organs. Every thrombosis which encroaches on the territory of this plexus will cause referred pain in these organs, and thrombosis spreading to the prostate or bladder would obviously be likely to result in disturbance of their function. Thus anal fissure cannot be considered a local disease, nor one *sui generis*, it is one result of the alteration of function which leads to the occurrence of these spontaneous thromboses in the region of the inferior hæmorrhoidal and

puddendal plexuses If the literature is searched for some explanation of the visceral symptoms just described, the rather vague and unsatisfying term 'reflex' will be found constantly recurring. When, for example, a patient with a fissure suddenly complains of a pain in the heel, this is described as a 'reflex' phenomenon. This is obviously merely a cloak for ignorance, and is no real explanation.

Let us now consider the significance of this particular symptom. Plantar pain is well known to be one early symptom of thrombosis in the lower limb veins. A probable explanation is that a small piece of clot travels in a retrograde direction (cf positive Trendelenburg's sign) and thus reaches a small radicle at the sole of the foot where it becomes impacted, causing acute pain. A thrombus in the pudendal plexus might reach the saphenous vein and ultimately be driven in the same way into a venule in the heel. This would account for the sudden occurrence of pain in this situation. Formation of thrombi, primarily in the pelvic veins (cf phleboliths) and their subsequent transport centrifugally into the saphenous system are, we believe, very common occurrences. They constitute an important part of the pathology of the rectal varicose syndrome.

The pain of anal fissure does not always come on in bouts—it may sometimes be continuous for hours on end. Nevertheless the onset is almost always determined by defæcation. Diagnosis of a fissure is not always easy, without anæsthesia and proctoscopy it may sometimes be impossible, for the ulcer is not always situated below the sphincter. The following is a useful aid to discovering the ulcer. The anterior and posterior commissures are palpated with a probe, if the fissure lies posteriorly, the palpation of the anterior commissure will be painless, and vice versa. In the great majority of cases, the sentinel pile serves as a guide to the fissure. We have already explained that this structure is the remains of a thrombosed 'external pile' after rupture—it is remarkable that this truth should have escaped the observation of even such an acknowledged authority as Bensaude. Close inspection of these polyp-like structures in the early stage will clearly show the necrotic opening through which the clot has emerged. The fissure itself is often the continuation of the open, gutter-like remains of the vein which formerly led to the varix in the 'pile'. The ulcer may assume various forms, it may be linear, or more oval in shape, a suppurating varix above the sphincter may produce exactly the same clinical picture as the more usual fissure at the mucocutaneous junction. The floor of the ulcer may

have a silvery-white appearance clearly recognizable as the exposed fibrous tissue of the vein wall (the intimal covering having been lost) In a newly formed fissure there may sometimes still be seen the orifice of a small vessel still oozing blood

A prominent symptom, not only of typical fissures but also of those similar ulcers which occur at a higher level, is the increased tone of the sphincter Because of this, inspection may at once suggest the presence of the ulcer, for the anus has a tightly indrawn, funnel-shaped appearance, and the sphincter offers noticeably increased resistance to the examining finger This response of the sphincter and corresponds exactly with that of other sphincters in the presence of ulceration or venous congestion Analogous behaviour is seen in the case of the œsophagus, cardia, pylorus, duodenum, jejunum, and bladder It is interesting to discuss the nature of this phenomenon

In German, the term "Krampfader" is often used for a varicose vein, and its significance is that such veins, wherever they occur, are accompanied by cramps of the neighbouring musculature Blood in a varicose vein flows more slowly than normal, so that some degree of stasis and congestion results This congestion will produce disturbances of function, the nature of which will depend on the organs affected Peller made an exhaustive study of the effects of experimental venous stasis on the circulatory system itself He was able to show that *venous stasis* produced an increase of pressure and alteration of the tone in the smooth muscle of the *arteries* These conclusions were drawn from studies of the shape of the arterial pulse-tracings together with readings of systolic blood-pressure The effect of stasis in a given area is not confined to that area, but is produced also on the smooth muscle of vessels at a distance He demonstrated this on patients suffering from cardiac asthma Temporary compression of the veins of one or both lower limbs led to a sudden increase of tone of the arteries of the whole body, and thus cut short even a severe crisis of cardiac asthma These at first sight rather contradictory results find an explanation in the work of Eppinger and Schwartz on the circulation in cardiac asthma This complex matter cannot be considered here in detail, Peller's vein-muscle reflex being mentioned only as in some degree analogous to the facts discussed in this chapter

Muscle reacts to venous congestion at first by increase of tone—by cramp, or spasm, such as is found with varicose veins Venous hyperæmia in the stomach, duodenum, or jejunum leads (as in gastritis,

duodenitis, or jejunitis) by irritation to spastic contraction of the muscles of these viscera, *cardiospasm*, *pylorospasm*, duodenal ileus, and ileospasm are the results. The same thing can be observed in the rectum and bladder—congestion in the rectal veins leads to hyperexcitability of the sphincter muscles. The spasm and cramp are not due to any ulceration that may be present, nor is the latter caused by the spasm, both alike are the results of the venous congestion. In other words, the congestion is primary, the spasm and the ulcer are secondary. Similarly in the case of the stomach, it is our considered view that so called gastritis is really a result of portal congestion and stasis.

If the congestion persists, the hypertonus of the muscles gradually gives way to a hypotonus and atrophy, and in many long standing cases of piles this can easily be demonstrated. The hypotonus is responsible for the soiling of the clothes and the rectal prolapse which tend to occur, this prolapse is, as it were, the final link in the chain of symptoms of the varicose syndrome. In recent fissures, the hypertonus of the sphincter can readily be appreciated by palpation with the finger, no attempt at further examination should be made without local anæsthesia. In more chronic cases the spasm may be overcome if a thoroughly greased finger-stall is used without undue discomfort to the patient, and without need for anæsthesia. A fissure lying at a higher level can then be palpated as a round depression. If a few drops of the quinine solution are applied to the base of the fissure at this first sitting it will begin to heal so that no anæsthetic will be needed for the second treatment. The symptoms of anal fissure may be simulated by high varices especially on the posterior wall by so called internal fistulæ, and by neuralgia of the anus. The character of the pains caused by internal varices is, however, a little different, they tend to be more constant and are independent of defæcation. The pain of an internal fistula can also be distinguished as a rule though we shall show later that internal fistulæ differ from fissures in situation only, and not in nature. Any attempt to make a rigid diagnostic rule from the character of the pain is doomed to failure. Anal neuralgia is extremely rare, we have seen only two examples among our whole series. Even in these the diagnosis was questionable. Both had been previously operated on for fistula and one of them developed three months later a recurrence of the fistula, after which his pain ceased. We have yet to see a case of genuinely idiopathic anal neuralgia.

THERAPY OF ANAL FISSURE

The non-surgical treatment of anal ulcers consists in regulation of the diet and bowels, and the use of suppositories and ointments. There is no doubt that these measures alone are sometimes successful. The good results of suitable diet and laxatives are due simply to the improvement in liver function, which relieves the chronic venous back-pressure from portal to caval system. In this way the factors which tend to produce varicose changes and thromboses at the anus are removed. This general therapy came into existence long before there was any deliberate attempt to understand its mode of action. The better understanding of the true nature of anal fissure merely provides an explanation for what was long known to be effective in practice. Indeed it may now be said that medical treatment properly applied is more rational than surgical intervention. To us the most valuable item seems the use of olive oil in the preparation of food. Other fats, especially butter and goose fat, are contra indicated. Constipating foods are to be avoided, and plenty of water should be taken. Locally, treatment by cauterizing the base of the ulcer under local anæsthesia seems rational, for in this way the remains of the vein are destroyed and the growth of healthy granulations made possible. The actual cautery may be used, being generally acknowledged to be quicker in its results than chemicals such as silver nitrate. Boyer was the first to propose division of the sphincter to heal a fissure. Recamier in 1838 introduced the method of massaging and stretching the sphincter. As this treatment often failed, Maisson-Neuve in 1864 advised forcible dilatation of the sphincter. His technique was to introduce the whole hand into the rectum, and then withdraw it with the fist closed thus tearing the sphincter. These methods have now been greatly modified, and the majority of surgeons to-day practise a slow and careful dilatation using the forefingers or an instrument. These procedures, however, seem to be less sound theoretically than purely medical treatment. It should be realized that the treatment of a fissure by stretching or tearing the sphincter and the laying open of a fistula with a knife are treatments based on a conviction that both lesions are really similar in nature, this bears out our own view that both lesions are basically and aetiologicaly identical—both are the result of thrombophlebitis. Some, for example Gant, have supposed that it is the stretching of the nerve-endings exposed in the floor of the ulcer which gives the relief. But

this is certainly not the true explanation. Shortly after Boyer published his method of dividing the sphincter, Copeland showed that simply nicking the muscle-fibres beneath the base of the fissure would give results quite as good as Boyer's severe procedure. Other alternatives are excision of the ulcer and suture (cf. excision of a fistula), and simple curettage of the ulcer (another method which may be used successfully for fistula). The common aim of them all is the destruction of the base of the ulcer, so as to allow the formation of healthy granulation tissue.

It must be clear that till now surgical treatment has been directed only at the one immediate symptom, the fissure. But since this ulcer is only a symptom, and the fundamental varicose disease remains unalleviated, the fissure is liable to recur if no other treatment is given. We have evolved a method which has the twofold object of promptly removing the symptom and then attacking its underlying cause. The destruction of the floor of the fissure, combined with 'vein-compression' therapy to bring to an end the reflux of blood from the portal into the caval system, seems to the author the logical procedure. The technique has been evolved as the result of both experimental and theoretical considerations. When, more than four years ago, the present writer rather hesitatingly abandoned operative for injection therapy (which had been advocated in Germany by Boas ever since the end of the last war), the method had as yet no scientific foundation. It was merely an empirical treatment borrowed from the English 'pile-curers' who a hundred years ago practised the method at fairs and markets in spite of the opposition of the medical profession. It is interesting that orthodox medicine after at first condemning the method, and later adopting it when the details became public property, has since tenaciously maintained all the original mistakes and errors of the pile-curers. It can well be understood that the latter, who popularized the method in England, America, and France, feared above all things the complication of suppuration, and for this reason excluded from their treatment fissures, fistulæ, peri-anal abscesses, pruritus, eczema with discharge, etc. These same contra indications are to-day, a hundred years later, *taken for granted by the profession without any critical examination*. Thus Anderson and Dukes, Dunbar, Scheffelaar, Klotz, Bensaude and Oury, Richter, Kirschen, Junghanns, and many others have issued warnings against injections in the presence of any inflammatory process in the rectum. Curiously enough, none of these

writers has sought to give reasons why eczema, fistula, fissure, pruritus, and abscess should be accounted contra-indications, or stated what would happen if the treatment were attempted in their presence. We decided to investigate this question, and the results were surprising because they directly contradicted the statements in the literature. We have shown that with suitable technique some of these 'complications' can be treated safely, and often cured in a remarkably short space of time. Fistula, fissure, and pruritus are in no sense contra-indications, but on the contrary are undeniably within the province of injection therapy.

As already noted our technique was at first rather primitive. Nevertheless, the successful results in the first hundred cases were astonishing. In these cases only two complications were encountered: one was the result of overdosage, which led to a periproctitis and ultimately a fistula, the other complication was a typical fissure artificially produced. It happened because at that time our endeavour, based on erroneous ideas in the literature, was to place the injection within the lumen of a vein in order to produce thrombosis. A small varix at the mucocutaneous junction was injected with a few drops of quinine solution and as a result clotted, necrosed and suppurated, finally becoming a typical fissure with all the usual accompaniments. With further experience



Fig. 12 Healing fissure

we gradually developed the ideas here described and in time came to use the following technique. The patient lies on his right side and the anal region is cleansed. The precise position of the fissure must next be determined, and the sentinel pile usually serves as a guide. If there is no external indication, the following method is helpful. A bit of cotton wool on a probe is dipped in 10-20 per cent cocaine solution and introduced into the anus. Pressure is now made carefully and steadily against first the anterior then the posterior commissure. After some minutes, the probe is cautiously removed. A bloody streak on the wool will indicate the position of the fissure. The fissure is now anaesthetized using about 5 c.c. of 1 per cent procaine solution. A fine needle is used, its point being entered in healthy skin and then passed deep to the fissure. If when anaesthesia is completed the patient is asked to bear down the

ulcer will open out and appear in its entirety. One or two drops of quinine-urethane solution injected in the base of the ulcer suffices to destroy the vein wall and permit the formation of granulation tissue (*Fig 12*). It is obvious that the dose of a chemical caustic is much more accurately controllable than is the actual cautery or the scalpel. With this method the sphincter is not interfered with, and the resultant scar is almost invisible. After the injection pain ceases, and the patient can usually resume his work at once. In 90 per cent of cases one treatment is sufficient for complete cure of the fissure, in the remaining cases two or three will be needed.

As the fissure has been made anæsthetic for the treatment, proctoscopy can usually be performed at the first sitting. Internal varices will invariably be found, and the 'vein compression' treatment of these may begin at once.

CHAPTER III

CONSTIPATION

ONE of the commonest manifestations of the rectal varicose syndrome is constipation. In cases with large varices and fissure it is almost always present. In pruritus and proctitis it may be absent. In these latter conditions, where the varices are of small calibre and chiefly in the sphincter region, the bowel habit may be normal. It is often found that in pruritus and proctitis the bowels may be opened two or three times a day. In most healthy people defæcation occurs only once in twenty-four hours, but two or three movements daily can of course be within normal limits, as well as only one stool in two or three days. The essential point is that healthy individuals respond to a defæcation reflex exactly comparable to the micturition reflex. In patients suffering from hæmorrhoids both these reflexes may be disturbed. We have gained the impression from many thousands of rectal examinations that patients with varicose disease do not experience a normal defæcation reflex. In these individuals, though digital examination may show fæces in the rectal ampulla yet the patient experiences no call to stool.

Constipation is, of course a symptom of many illnesses. It occurs in gastric and duodenal ulcer, in liver and gall bladder diseases, and in heart disease where there is liver congestion. Fleiner considered the constipation to be due in some way to bowel spasticity. We have already pointed out that venous congestion and spastic hyper excitability go together. Thus venous stasis affecting the bladder or rectum produces increased tone of the respective sphincters, which in the case of the rectum can be verified by direct palpation. Our work has led us to the belief that the prime cause of the rectal varicose syndrome with all its varied manifestations is a stasis of blood in the portal system. Wherever this stasis exists, whether the cause be central or peripheral, there will tend to be some degree of constipation. Moreover, if our conception is accurate, it becomes easy to understand how blood will be driven into the pudendal and inferior hæmorrhoidal plexuses and by causing congestion there will interfere with micturition. And in fact periproctitis

(thrombophlebitis purulenta acuta) and anal fissure (thrombophlebitis marginalis recti chronica) are often accompanied by dysuria. The simple removal of a small thrombotic nodule may lead to a disturbance of micturition lasting several days. In order to understand the pathology and logical therapy of constipation, an analysis must be made of all the factors which experience has taught us can produce it. In this way we may test the hypothesis that there is really one common cause—namely, some impediment to the flow of portal blood to the liver leading to a partial backflow into the caval system.

All will agree, for example, that the type of food eaten, and the amount of fluids taken, influence the bowel action, and it is clear that pressure conditions in the portal circulation must be largely dependent on these factors. Bile retention produces constipation and here again it is easy to understand how close a relation there must be between the bile flow and the portal pressure conditions. Where the obstruction is in the liver itself, the whole inferior cava system may be affected in addition by pressure upon, or distortion of, the vena cava as it passes through the diaphragm (*see Chapter VIII*). As far as heart and lung diseases are concerned this would explain the constipation they produce. In the majority of my patients with so-called 'habitual' constipation, where no systemic cause could be discovered with the diagnostic aids at present known, large rectal varices were present. It is quite likely that these patients were really suffering from some minor degree of portal stasis of which the constipation was the earliest manifestation. Many who are labelled hypochondriac and neurasthenic and complain of constipation, reveal themselves as suffering from piles if a really thorough rectal examination is made. Their nervous symptoms will disappear when the portal reflux has been prevented by injection and 'vein compression'. It is indeed remarkable how the nature of these patients changes, but this is not the place to discuss the possibility of effects upon the mind of toxic substances from the portal circulation being released into the general blood-stream. Constipation from mechanical causes such as stenosis after ulceration, bowel paralysis, and so forth is of course excluded from the present discussion. In comparison with the so-called 'spastic constipation' their occurrence is very infrequent, indeed almost negligible—our own figures suggest about a hundred to one. In large towns at any rate 'habitual' constipation is the commonest of all human ailments.

The chief symptoms of this constipation are headache, yellow

complexion (sometimes almost icteric), periods of depression, and very often cramps of the sphincter ani and the levatores ani muscles. In the later stages atrophy of these muscles and a tendency to prolapse are found. An enormous amount has been written on the treatment of this condition, and the aperients, irrigations, and dietetic cures that have been advocated are without number. Medicines very soon lose their effect, and they have no curative value. Even operative methods have been tried, perhaps the commonest having been dilatation of the sphincter ani.

As soon as we had treated our first hundred cases with injections we became impressed with the fact that many who had had to take laxatives for years were now relieved of their constipation, and we have since given special attention to this symptom. A simultaneous disappearance of the constipation and the varices and sphincter spasm has been noted in hundreds of cases. Among these were patients who since their early youth had needed to take purgatives. One, a woman of 70, had for thirty years never obtained a single bowel action without wash-outs and aperients, yet she became free from constipation. Another woman of 51 gave the following history —

Even twenty years ago I was constipated. I always took medicine, but without getting the effect I desired. Sometimes the result was diarrhoea which interfered with my work. One day I decided to try an enema. This was successful in so far as by spending a quarter of an hour in this way I was comfortable for the rest of the day. I therefore continued to use enemata, but after about a year I found I needed two or even three daily. Recently, twenty years after I began the practice, it required 15 to 20 a day to secure any result. I took them regularly at the appointed times without ever feeling any desire for defæcation. A good two hours of my time was thus occupied each day. But this was not my only trouble. I sometimes had severe itching, and at times there was a discharge from the bowel. On two occasions there was severe bleeding. Since the treatment I have had a normal daily bowel action without the need for any artificial aid whatever. I can now eat whatever I like, whereas for years before I had eaten practically nothing but green vegetables and fruit.

Hæmorrhoid patients who are constipated nearly all have the same experience after treatment. Proctoscopic examination in these cases reveals internal varices of truly astonishing size, sometimes veritable tumours of blood-vessels. We believe that these tumour-like swellings act as a mechanical obstacle to defæcation. But the chief hindrance is the hypertonus of the sphincter due to portal congestion. The response of the constipation to 'vein compression' therapy strongly supports the views expressed in this book.

CHAPTER VIII

SPONTANEOUS THROMBOSIS IN THE RECTAL VEINS—
 LOCAL AND GENERAL PRURITUS—
 THE PROBLEM OF EMBOLISM

THE literature of proctology makes little reference to spontaneous thrombosis or to pruritus ani. The latter is considered to belong to the province of the dermatologist, and dermatological writings contain a confusion of theories about the causation and treatment of the complaint. There is probably no method of treatment which has not been tried for this very common trouble, and it is obvious from the multitude of salves, suppositories, mixtures, powders, and pastes advised that none can be very effective. Upon the aetiology of general pruritus ideas are no less chaotic.

It has long been recognized that pruritus ani and hæmorrhoids occur together. Many authors regard mucosal polyps, fissures, fistulæ, mucosal prolapse, proctitis, and cryptitis (all by them considered to be independent diseases) as potential causes of pruritus ani. As for general pruritus, nearly all known diseases of the abdominal organs have been inculcated. In particular, liver insufficiency and hepatic diseases such as hepatitis, cirrhosis, carcinoma, gummata, hydatid cysts, jaundice, and cholelithiasis may be mentioned. Diseases of the stomach, too, chronic appendicitis, colitis, as well as diseases of metabolism, leukaemia, genital conditions such as ovarian tumours, pregnancy, menstruation, prostatic and vesicular troubles, malignant tumours of the true pelvis, constipation, and malaria are all on the list. In America a search has been made for some specific exciting substance.

A critical study of the literature suggests two generalizations —

1. Although it has been demonstrated again and again that a common specific cause for all cases is unlikely, the idea of such a possibility still lingers.

2. A survey of the diseases associated with pruritus shows that the organs affected are those connected with the portal circulation, or those which may in certain circumstances (as we shall endeavour to show) come to have this connexion. Tumours above the

diaphragm, and diseases of the thorax, head, neck, and upper limbs are scarcely ever associated with pruritus, either local or general.

In an endeavour to simplify the problem, writers have classified pruritus into two morphological types—distinguishing the sharply localized cases—pruritus ani, scroti, vulvæ, cruris, and inguinalis—from the generalized. In the latter group, juvenile, senile, climacteric, menstrual, pregnancy, and summer and winter types have been described. In plain English, these terms merely mean that generalized pruritus may occur in young and old of both sexes, and that it may be related to menstrual, pregnancy, seasonal, and climatic changes.

It has long been known that pruritus is in some way connected with varices. It is a well-established fact that healthy young women during pregnancy may develop hæmorrhoids and varicose veins. That itching also may make its first appearance at this time is equally well known. In seeking the cause of pruritus, one must pass in review all those disturbances which may be associated with portal or caval venous congestion. Thus nutritional upsets which may lead to liver congestion, and all derangements of the abdominal organs from which the portal blood is collected, have to be borne in mind. Circulatory affections also have to be considered in so far as they may embarrass the portal system.

In the literature, spicy foods, tea, coffee, strong alcoholic beverages, and constipating drugs are mentioned as causing pruritus. In all these cases it is still to be determined how far the substance in question may impede or disturb the return of blood from the bowel. And since perineal irritation is common in diseases of the female genitalia—the bladder, prostate, and testicle, some local change in the circulation must be looked for in all these conditions.

In order to get to the bottom of the problem, every observation which appears to be soundly based on fact must be taken into account. It is for instance quite certain that pruritus has favourite sites, and that these are in the areas affected by varicose veins. It is also certain that on the upper limb and on the trunk above the diaphragm localized pruritus is so rare as to be negligible. Moreover, the changes with which it is associated do not extend to the ampulla of the rectum, no eczematous patches being ever found there or in the colon. In over 680 cases of pruritus of the anus, vulva, and scrotum, we have never seen the mucosa above the pars sphincterica involved. This suggests that the changes do not occur above the limits of the

inferior hæmorrhoidal plexus. Since this is part of the caval system it is right to say that localized pruritus occurs exclusively in regions drained by the inferior vena cava. The significance of this fact is that *pruritogenic properties have been ascribed to certain metabolic breakdown products which are readily absorbed from the bowel*. The liver in the ordinary way detoxicates these substances, hence the liver and its afferent vessels must possess a certain degree of immunity to their action. The inference is, that these substances only lead to pruritus when they escape into the general circulation by way of varices which connect the portal and caval systems.

In the last five years, several thousand proctoscopies have been carried out by the author, and as a result certain observations have been made which may perhaps throw some light upon this obscure subject, and incidentally on the problem of embolism. Spontaneous venous thrombosis is a phenomenon which occurs more commonly in the inferior hæmorrhoidal zone than anywhere else in the whole body. Day after day patients of all ages and both sexes have been seen with spontaneous thromboses varying in size from a pinhead to a plum, both at the anus and in the rectum above. These sudden thromboses may sometimes occur in apparently healthy young people without warning. They are very painful and until now it has not been customary for surgeons to examine such patients proctoscopically for the following reasons —

- 1 Their pain is such that they cannot tolerate this examination as ordinarily carried out. even digital examination is in many cases unbearably painful.

- 2 At this stage the wrong diagnosis of 'incarcerated internal piles' is generally made, and the trouble is treated with hot applications and baths.

- 3 With the proctoscopes in general use examination if performed may fail to disclose any abnormality. A side window proctoscope such as the author's, is particularly well adapted to the exact examination of these cases. In almost all of them high lying varices at the level of the superior hæmorrhoidal plexus are found to be present.

At this point it is convenient to turn to the subject of thrombosis and embolism. The thromboses artificially induced in the treatment of varicose veins of the leg are fundamentally different from the spontaneously occurring thromboses in the inferior hæmorrhoidal plexus. With the former type, as also with that occurring in the

neighbourhood of infections, embolism is relatively rare. Research on the subject of thrombosis has concentrated on (1) changes in the blood, (2) changes in the vessel wall, and (3) slowing of the blood-stream, and these three factors have been looked upon as entirely controlling the occurrence of thrombosis. Chemical irritants are used to damage the vein wall when it is desired to produce an artificial thrombosis. In this manner it is possible to cause the clotting at whatever point in the vein may be desired.

In spontaneous thrombosis, on the other hand, there are certain 'sites of election' at which clotting is common. If it is granted that the three factors mentioned above are causes of thrombosis, it still remains to find an explanation for these sites of election. What, for instance, makes this strictly limited zone at the anus the commonest site in the whole body for spontaneous thrombosis? Were 'blood changes' really the determining factor, we should expect clotting to occur wherever there are varicose veins and consequently a sluggish circulation, but this is not the case. Because a patient has had a spontaneous thrombosis at the anus, it does not follow that it is particularly easy to induce thrombosis in any other varicose veins he may possess, although any blood changes are presumably present there also. Perhaps it is in this very assumption, that the same blood changes are present in all the varicose veins of a patient, that the fallacy lies. It may well be that an important clue lies hidden in the fact that these sites of election undoubtedly exist.

In a paper read before the Gesellschaft der Aertze in Vienna in June, 1934, the author drew a comparison between varicose disease of the lower limbs and anorectal varicose disease. Thus for example, pruritus and eczema ani were compared with eczema and pruritus of the shin. The general analogy is enhanced by the fact that in these two regions spontaneous thromboses most commonly occur. Now one of the statements most frequently made by sufferers from pruritus is that the itching (both in anal and in varicose vein cases) tends to come on and become really intense on retiring to bed at night, the interference with sleep is often a serious part of their troubles. The literature, and especially dermatological literature, explains this as being due to the warmth of the bed. This seems to us an inadequate explanation, indeed many patients find that a really hot bath is the only way to secure relief. Moreover, the temperature of the anal region in a fat individual sitting in an overheated office in the winter is surely no lower than when he is in bed.

Yet his itching is definitely worse at night. If the warmth really were responsible an ice-bottle or a cold bath would be an effectual remedy, but such treatment is almost always useless. Can the true explanation lie not in the warmth of the bed, but in the horizontal position? Here a further important analogy with the lower limb may be drawn. Trendelenburg's sign, as is well known, is elicited as follows: the patient lies in the horizontal position, and the varicose veins are emptied by raising the limb above the level of the body; a tourniquet is now applied to the thigh, and the patient is allowed to stand up. It will be found that the previously distended veins remain empty. Again, if a tourniquet is applied to the patient in a standing position, while the leg veins are distended with blood, and if he then walks to and fro, the veins will be found to empty themselves and remain collapsed. These long-accepted facts show that the direction of blood-flow in such a patient when upright is not upwards in the varicose veins but downwards, i.e. centrifugally. Under these conditions, some blood from the pelvic veins will find its way into the saphenous system. On the other hand the same tests show that when the leg is lifted above the horizontal the blood flows centripetally in the same veins and the whole saphenous tract drains into the veins of the pelvis. In other words, when varicose veins are present, a change of position on the part of the patient may lead to reversal of flow in the affected veins. Morro has shown that the deep veins may be filled by injecting fluid into the subcutaneous veins, and vice versa. The occurrence of the reversal of flow was demonstrated by Magnus to the Deutsche pathologische Gesellschaft in 1921.

The present writer has observed an exactly analogous train of events in the case of the hæmorrhoidal veins. In many patients with piles, and in all cases where fissure or perianal thrombosis has occurred, *the direction of blood-flow is capable of reversal*, being sometimes from superior to inferior hæmorrhoidal plexus and sometimes in the opposite direction. If such a patient is asked to strain down, it becomes evident that an increase of the intra-abdominal pressure (such as occurs also during defæcation) causes the inferior hæmorrhoidal veins to become distended with blood from the valveless veins of the superior plexus. Only a free reflux of blood from the portal system could explain the profuse bleeding of patients with piles during defæcation, bleeding which may give rise to the gravest anaemia. And this same reflux, occurring under

certain conditions of pressure, offers a most reasonable explanation for anal pruritus and thromboses

The question why pruritus and thrombosis are so commonly observed in this localized area, demands an answer. Our reply is, *that this is just the place where portal and caval bloods intermingle, more especially under pathological conditions*. A review of some anatomical points may be helpful at this juncture

The rectum has the following arterial supply —

- 1 The unpaired superior hæmorrhoidal artery, from the inferior mesenteric

- 2 The right and left middle hæmorrhoidal arteries, from the respective internal iliacs

- 3 The right and left inferior hæmorrhoidals branches of the respective internal pudic arteries

The first of these is chiefly concerned with the ampullary part of the rectum on the dorsal aspect of which it divides into two main branches. Their derivatives anastomose with those of the inferior hæmorrhoidal arteries, which supply the anal area only

The rectal veins (Fig 13) are as follows —

- 1 A single superior hæmorrhoidal vein

- 2 Right and left middle hæmorrhoidal veins

- 3 Inferior hæmorrhoidal veins several in number, and branches of the sacral venous plexus

Of these, the superior hæmorrhoidal vein is valveless, and drains into the inferior mesenteric vein, which in turn empties into the the splenic and finally the portal veins. The others possess valves and drain into the hypogastric and pudendal plexuses. All the rectal

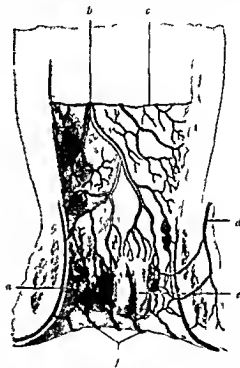


FIG 13. Semi diagrammatic view of the veins of the rectum showing the venous anastomoses of the annulus hæmorrhoidalis. *a* Venous anastomoses. *b* Superior hæmorrhoidal vein. *c* Cutaneous membrane. *d* Middle hæmorrhoidal vein leading to pudendal plexus. *e* Column of Morgagni. *f* Inferior hæmorrhoidal veins. Based in part on the work of Marfan. *Thèse de Lyon* 1893. (*Cornu*)

veins are intimately connected to form the hæmorrhoidal venous plexus. In the middle hæmorrhoidal region what might be described as a watershed is found, it is the boundary zone between the portal and caval systems, which are here in close relation with one another. The middle hæmorrhoidal veins lie between the two diverging streams. Portal blood differs fundamentally from that in the general venous circulation in that it contains metabolic products from the bowel and toxic substances which in normal circumstances are dealt with by the liver and never reach the general circulation. *If, however, a reflux of blood from portal to caval system takes place, a change in composition of the caval blood must result.* This locally produced adulteration of the blood provides a much more satisfying explanation of the localized type of pruritus than does the hypothesis of some generalized blood impurity. In the case of the leg we have already described how a change of posture may reverse the direction of venous flow and if the horizontal position may reverse the flow in the saphenous system presumably changes must occur also in the pressure conditions in the veins of the pelvis. It is known that pressure upon the inferior vena cava as from a tumour, will give rise to the development of collateral circulation which passes by way of the epigastric and internal mammary veins to the superior vena cava (producing the so called caput Medusæ). There exists a similar free anastomotic network between the abdominal and pelvic veins and the gluteal vessels, and thus with the saphenous and femoral systems.

It is, moreover, recognized that hepatic cirrhosis or metastases or a tumour at the hilum of the liver, will lead to a portal stenosis the effects of which extend right back to the ultimate capillaries of the system, and give rise to what are known as 'secondary' piles. In this connexion it should be noted that even under physiological conditions there are fluctuations in the portal blood pressure. Regelsherger has investigated the question of the rhythmic variations in the liver's activity. According to this worker, the liver is the most important source of the body heat. Forsgreen also deals with this, and shows that fluctuation of the temperature of the body corresponds with the rise and fall of the liver's activity. The periodical changes in the level of the blood-sugar, and in bile- and water-excretion by the liver, are also well known. Another example of periodicity is the sensation of hunger, which appears at definite times and lessens if the normal meal-time is allowed to pass. Now

as the activity of the liver varies, the pressure in the portal system will alter. As these changes must be transmitted to the hæmorrhoidal plexuses, it follows that the itching of pruritus ani may well depend upon these same changes. Proctoscopic examination of a case of piles at various times in the day will show that the hæmorrhoids vary in size. The difference in the degree of distension of the varices before and after defæcation is also most striking.

It seems that the greater the amount of metabolites and toxic substances present in the portal blood, and the higher the pressure in the portal system, the more intense the itching will become. This explains the periodic attacks of itching. It also explains why morphia does not relieve the irritation, for the action of the drug is on the central nervous system, while the irritating effects of the toxins are conveyed by the sympathetic system accompanying the vessels. For this reason the itching may be present even during deep sleep. It is very probable that the horizontal position favours the reflux of portal blood into the caval system, for it is reasonable to suppose that the greatly diminished pressure in the inferior vena cava which must result from the horizontal position will allow portal blood to enter the system, following the line of least resistance. It is the author's opinion also that the skin changes associated with varicose veins of the leg (erythema and eczema cruris) may similarly be the result of a retrograde flow of portal blood into the pelvic veins and finally downwards into the saphenous system. We may now group the factors upon which pruritus depends under the following heads—

- 1 Generalized pruritus will appear if portal blood reaches the general circulation by passing through the anastomoses which connect the two systems

- 2 The favourite sites for local pruritus are found in those areas where the systems adjoin—anus, perineum, scrotum, vulva, inguinal region, and lower limb (*Fig. 14*)

- 3 Where no anastomoses exist, as for example in the upper limb, localized pruritus does not occur

- 4 The degree and intensity of pruritus will depend upon (a) the freedom of the anastomoses between the two systems, (b) the degree of congestion in the portal vein, (c) the type of food taken (which will influence the composition of the portal blood), and (d) the degree of digestive activity

The author has been impressed by the fact that pruritus is much less common in patients who bleed from their varices. This may

perhaps be because these patients eliminate the pruritogenic substances by the bleeding, thus keeping the inferior hæmorrhoidal zone free from their action. Bleeding is a feature of large and medium-size varices, whereas in pruritus the minute veins are chiefly affected. Two types of 'piles' patients may be distinguished—those with large and medium-size varices comparable with those of the knee region and those with small dilated venules, like those on the dorsum of the foot and over the malleoli. It is principally in this latter type, with

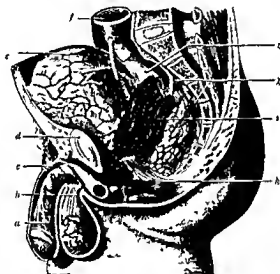


Fig. 14.—Veins of the male pelvis showing anastomoses between the veins of the scrotum and the hæmorrhoidal plexus. *a* Corpus cavernosum urethra; *b* testis; *c* scrotal vein; *d* symphysis pubis; *e* peritoneum; *f* sigmoid colon; *g* rectal ampulla; *h* internal iliac artery and vein; *i* venous plexus; *k* internal pudendal artery (Corning).

tiny varicosities in the inferior hæmorrhoidal zone, that pruritus occurs. In these cases the mucosa in the sphincteric region is affected in the same way as the anal skin—it is reddened, succulent, and redundant, or œdematous, there are breaches in the epithelial covering. We consider all these changes to be the result of chronic poisoning by substances in the portal blood. Erythema cutis ani and 'proctitis' are analogous lesions produced by identical causes. The portal blood with its toxic contents reaches not only the anal zone, but passes also by way of the pelvic plexuses to the genitalia and even the

saphenous system. In this way there may arise a chronic intoxication of the perineum, the scrotum or vulva, the genital organs, and the lower limb. Mucosæ cannot give the sensation of itching, but the affected areas ooze, and this may possibly explain some cases of vaginal discharge—leucorrhœa—which have hitherto remained inexplicable. Varicose lesions of the leg—erythema, dermatitis, and ulcer—may be similarly explained. The important fact that the lesions do not occur on the upper limb is simply due to the impossibility of access of portal blood to that part. If the mucosa in anal pruritus is studied proctoscopically, it will be found œdematous and thickened, with little raw areas, and a tendency to bleed easily. Instead of the normal pinkish colour, it has a dusky or bluish appearance. If the low-power lens of the proctoscope is brought into use, a veritable tangle of dilated venules will be seen. Now this is precisely the description of '*chronic proctitis*', a condition the aetiology of which has always been obscure. If we exclude the extensive coloproctitis which is associated with bloody and purulent discharge, diarrhœa, and tenesmus, there remains a group of '*proctitis*' cases of chronic type, which we now believe to be part of the varicose syndrome of the rectum, and which is almost always found with anal pruritus and eczema. The changes are identical with those in the anal skin described as *erythema cutis ani*.

In the author's view, proctitis in patients with hæmorrhoids, not extending above the rectum, is totally distinct from the more extensive coloproctitis. The aetiology of the latter disease is unconnected with the rectum and therefore does not concern us here, but with regard to the former type, the localized chronic proctitis, the author has come to the firm conclusion as a result of his observations that *the proctitis, erythema, eczema, and pruritus form together a definite clinical entity*. This entity is one part of the rectal varicose syndrome, and is simply the expression of the irritating effect of the portal blood on the lowermost parts of the rectum and the anus. Strong support is given to this conception by the improvement in all the symptoms and signs which follows 'vein compression' injections. This type of proctitis seldom extends up into the ampullary part of the rectum, because the portal vessels are immunized. The toxins do, however, damage the anal mucosa and skin, and, as we now believe, the skin of the leg where Trendelenburg's sign is positive. If spontaneous thromboses, too, are due to the same fundamental cause, namely, the reflux of portal blood, this raises the question why sometimes

thromboses occur, and sometimes pruritus. Any one examining many of these cases will agree with the following observations. Thromboses may occur in any *varix* at the anus, whether of small, medium, or large calibre, pruritus occurs where the varices are of the small type. Again a comparison may be made with the lower limb, where thrombosis of large vessels is almost exclusively confined to the thigh, the knee, and the upper third of the leg. Pruritic skin changes, however, and ulcer, are found in the region of the smaller varices about the malleoli and on the lower two-thirds of the leg. The distinction would seem to be that portal reflux occurring suddenly as the result of some sudden change of conditions tends to cause acute dilatation of previously formed varices, resulting in thrombosis. A more chronic, slighter reflux, especially where the blood is brought into close contact with the skin, as in the lower part of the saphenous tract, is responsible for the itching and skin changes. The same would apply to the upper and lower parts of the hæmorrhoidal region respectively. *Pruritus and thrombosis differ only in degree, both are symptoms of differing degrees of the same functional disturbance.*

The pathological anatomy of varicose disease of the lower limbs so closely resembles that of the rectum that it is natural to look for a cause common to both. The only discoverable common factors are (1) their proximity, (2) the intimate relations of their vascular systems and (3) the possibility of a pathological reflux of portal blood reaching both regions. Such a conception renders the whole picture of spontaneous thrombosis, its sudden onset and its symptomatology, much easier to comprehend. It becomes obvious that the regions where the two venous systems meet must necessarily be areas of predilection for thromboses, and wherever the portal blood may find its way under conditions of backflow thromboses will occur, as, for example, in the pelvis and the lower limbs (but seldom if ever in the arms).

If thrombosis occurs in an artery, the affected region loses its blood-supply, and can only be nourished through anastomotic channels. With these veins, on the other hand, thrombosis of even a large trunk need not necessarily produce stasis (and therefore œdema). This is because the blood in these veins is flowing centrifugally, and therefore plays little part in the venous return from the area which they drain. In this sense the thrombosis of a large vein such as the saphenous may be taken as an attempt at natural cure, for the clot acts as a ligature preventing access of the portal blood to the vessels beyond. It is interesting to note that the places where thromboses

occur naturally are found by experience to be the best places for ligaturing the veins. In exactly the same way, spontaneous thromboses at the anus may be looked upon as curative.

The anastomosis of the superior with the inferior hæmorrhoidal plexus is intended to act as a dam against the downward flow of portal blood—for the channels unless pathologically dilated must offer considerable resistance. A consideration of the question of embolism will help to indicate the importance of this function. Embolism occurs when a thrombus is set free and carried by the blood-stream until it is held up in a vessel too small to permit its passage. Thus embolism implies previous thrombosis. If we consider the commonest form of thrombosis, that is, spontaneous thrombosis, in contradistinction to septic and artificial thromboses, we may obtain a clue to the problem. From what has been said already it will be clear that in our view the *essential factor in producing spontaneous clotting is the centrifugal flow of portal blood into the caval system*. The chain of events may be summarized thus: (1) Portal stasis and congestion, (2) Varicose dilatation of the portal radicles, (3) Back flow of portal blood through these channels.

In the pelvic veins the thrombi which tend to form may remain in the ample venous plexuses without in any way embarrassing the venous circulation. In course of time they may become absorbed with recanalization of the vessel, or they may become calcified. That this latter is a common happening is shown by the frequency with which phleboliths are to be seen in radiographs of the pelvis. Should the thrombus, however, become detached it may be carried by a reflux of blood back into the saphenous circulation until it reaches a small vessel which it completely fills. On the other hand, such a thrombus (unlike the septic and artificial thrombi, which adhere firmly to the vessel wall and seldom or never become detached) may be carried into a main vessel such as the saphenous trunk and thus find its way into the general circulation. Only some such conception as this can explain the observed facts.

Once these ideas are accepted several otherwise incomprehensible facts are explained. Payr, for example, mentions that commencing thrombosis in the pelvic veins may be associated with the sudden onset of hæmorrhoidal symptoms or anal tenderness.

The reason why the incidence of embolism is high after pelvic operations now becomes easy to see. This applies especially to gynæcological operations, where the application of mass ligatures to

the venous network in the broad ligaments is a potential source of danger because any sudden increase in the portal pressure is readily transmitted via the dilated pudendal plexus, and may cause an insecure ligature to give way. Precautions against such a risk should be taken in all operations within reach of the portal system. Prostatectomy is especially dangerous in this respect. The terrifying hæmorrhages which may occur after this operation are known to all surgeons. Even after the most careful hæmostasis such a disaster may occur—the

blood coming from the portal system. Bleeding is particularly liable to follow the first defæcation, the effort causing a rise of pressure in the portal vessels, so that blood is forced through anastomotic channels notably the middle hæmorrhoidal plexus. If the anastomoses are enlarged i.e., the vessels varicose the resultant hæmorrhage may be very severe.

Many points in the symptomatology of pruritus ani have hitherto lacked explanation. The anal skin in this condition is reddened, swollen and moist, and later, radially arranged oedematous folds appear (Fig 15). The skin tends to lose its pigment, and here and

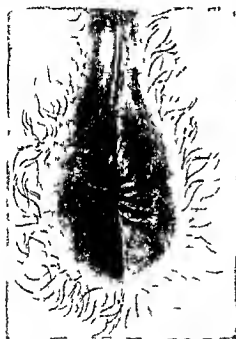


Fig 15.—Pruritis ani with erythema ani

there the epithelium is deficient. Finally, the skin becomes pale, cracked, and atrophic. The sphincter passes from increased to diminished tone. It is interesting to observe that during treatment as the itching diminishes there is a return of the normal pigmentation of the skin.

It has not yet been decided whether the itching sensation travels by the spinal nerves or by the autonomic nervous system. That stimulation of the autonomic fibres to the skin need not cause itching

is obvious, because vasodilatation (flushing, amyl nitrite) and vasoconstriction (Raynaud's disease, adrenaline) are not associated with itching. The question whether the itching or the skin changes come first may be answered quite definitely as far as senile pruritus is concerned. In this disease, the itching precedes the skin changes. This may be compared with urticaria, where the itching may have passed its maximum intensity before the wheal appears, indeed it may have ceased. The generalized pruritus which comes on in patients from 60 to 80 years of age occurs in attacks, and tends to be worse at night. In these cases a sub-icteric tinge may be noticeable, suggesting some liver damage. The generalized pruritus of younger people runs a similar course—these patients also stating that the itching is aggravated at night. Secondary pigmentation is a characteristic of both types of pruritus.

It has been shown that certain peptones if introduced into the skin are capable of producing itching, it is therefore conceivable that albuminous breakdown products of metabolism, if they succeed in reaching the general circulation in high concentration, may have a similar action. The 'alimentary' type of pruritus, associated with tea- and coffee-drinking is well recognized. In local pruritus the toxins only reach sufficient concentration at the actual place where they enter the general circulation. When they reach the larger veins increasing dilution renders them innocuous. The intermittent nature of the pruritus also fits in well with this hypothesis, for only when digestion is at its height and portal congestion is increased will the toxins escape in sufficient quantity to produce symptoms. For the pruritus to become generalized incomparably greater quantities of toxins must obviously enter the general circulation. It is possible that in these cases the metabolites may enter the circulation in other ways than via the hæmorrhoidal plexuses, at any rate the possibility merits consideration.

According to Frerichs and Lanceraux, pruritus occurs in only one-fifth of all cases of jaundice. Prostate and bladder troubles may also be associated with pruritus (and senile pruritus often disappears after prostatectomy). Pruritus associated with menstruation closely follows the ovarian cycle. That seen at the menopause is probably nothing more than the pre-senile type occurring in the female. Menstruation and pregnancy types are usually generalized, but we had the opportunity of observing one case where itching was localized to the groins and became worse with each menstrual period. Since not

every patient with icterus experiences itching, and since the intensity of the jaundice is independent of the degree of pruritus (the icterus can disappear without cessation of the itching) it follows that the pruritus is independent of bilirubin concentration in the blood. In other words, the itching does not wax and wane with the rise and fall of blood-bilirubin. Similarly, in diabetes we have seen the blood-sugar return to normal with insulin without any effect on the pruritus. Hence it would seem that neither the sugar nor the bilirubin is in itself pruritogenic. The substances actually responsible need not necessarily correspond with the sugar or bilirubin in their concentration. In localized pruritus these substances, as we hold, derive from the portal blood, and in generalized pruritus they may well have the same source. An analysis of the various conditions in which generalized pruritus may occur will show that there is common to all the possibility of some liver disturbance. This disturbance may be central, that is, the liver itself may be affected and act as an obstacle to normal portal blood-flow, or it may be peripheral, the obstruction being in the vena portæ or its tributaries. Pruritus is very commonly observed in the terminal stages of malignant abdominal tumours, this is due to the liver metastases interfering with the flow of portal blood. Other examples of hepatic causes are hydatid disease and cirrhosis, liver gummata, acute hepatitis, and cardiac insufficiency with secondary hepatic damage.

Tumours of the head of the pancreas which compress the portal vein are an example of a peripheral cause of portal stasis. The tumour need not necessarily be malignant, acute or chronic pancreatitis, shrinkage of the pancreas, an inflammatory glandular mass at the liver hilum, and tumours and cysts of the kidney can all exert direct or indirect pressure on the portal vein, making possible a periodical reversal of the flow of blood within it. Stones in the common bile-duct occasionally lead to pruritus in the same way. Such a stone may cause, of course, at the same time a rise in blood-bilirubin and (by direct pressure on the vein) a rise in portal pressure. It has long been known that 'secondary' hæmorrhoids may result from liver tumours, but it has never been realized that in fact all hæmorrhoids are secondary. The entire rectal varicose syndrome is dependent upon some cause external to the rectum. There is really no essential difference between piles which are due to some abdominal tumour and piles which are apparently idiopathic. Sometimes the underlying cause is overwhelmingly important (as, for example, an abdominal

carcinoma), while at other times it is relatively insignificant (e.g., chronic hepatitis), yet the pathological process is the same in both cases.

It is easy to take the view that mechanical compression of the portal vein by the gravid uterus may be the cause of pruritus in pregnancy. Indeed, the pressure of the uterus is acknowledged as a cause of varicose veins of the leg. Due consideration must, however, be given to these important objections to such a view. (1) The varicose veins may begin to appear almost at the beginning of pregnancy, that is, before enlargement of the uterus can play any important part. (2) In spite of the large uterus of advanced pregnancy, varicose veins do not occur in all cases. One of the earliest signs of pregnancy is the bluish discoloration of the vulva, which points inescapably to the occurrence of some profound change in the circulation in the pelvic veins. Indeed, it is hard to explain the discoloration and obvious enlargement of the vulval veins otherwise than by some interference with the drainage of the pelvic veins. The very great development of the uterine and pudendal plexuses during pregnancy is well known. Yet in spite of this increase in volume of the veins (which drain also the vagina and vulva), there is at the same time increasing congestion and stasis in their radicles. Why should this congestion arise before the uterus is notably enlarged?

In order to explain these changes taking place in the veins of the genital organs we have to admit that some factor hitherto overlooked is operative. Normally, the blood from these organs is received by the inferior vena cava (*Fig. 16*). Now, though the circulation of the fœtus is anatomically distinct from that of the mother, the fœtal waste products are brought in the umbilical vein to the placenta and transferred to the placental vessels of the mother. Thence the products must be carried to the vena cava and the general circulation if the anatomical considerations in the diagram are correct. How does the mother deal with these toxic substances? The poisons from her own alimentary tract are taken to the liver which is the great chemical works of the body and there rendered harmless, it is hard to believe that no provision is made against the flooding of the maternal circulation with toxins from the fœtus. It is much more probable that at the beginning of pregnancy the pelvic venous system becomes altered, the anastomoses between the portal and caval portions enlarge and blood from the genitalia tends more and more to pass into the portal system, carrying with it the fœtal toxins. The idea of such a diversion

of blood is not new, it occurs in varicose veins (positive Trendelenburg sign), in varicoceles and hæmorrhoids, and in the caput Medusæ which develops when the caval return is obstructed by a pelvic tumour. In the case of pregnancy, such a diversion would effectively prevent the foetal toxins from doing any harm to the mother. It may throw

new light on the toxæmias of pregnancy, for if the change does not take place or is imperfect, the resultant disturbances would presumably be just such toxic manifestations as are well known to be associated with pregnancy.

Perhaps the discoloration of the external genitalia at the beginning of pregnancy signifies that the anastomotic communications are not yet fully opened up. The small communicating channels can not yet accommodate all the pelvic venous blood, with the result that the small vessels of the vagina, uterus, and broad ligament become congested, some of the blood still finding its way to the vena cava (Fig 17). This would explain the toxic symptoms sometimes noted in early pregnancy. It is certainly striking that if the pregnancy is interrupted the symptoms at once vanish. This line of

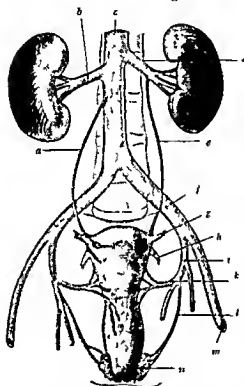


Fig 17.—Showing the venous drainage of the veins in the uterus: 1. the inferior vena cava; a. Left ovarian vein; b. Right renal vein; c. Inferior vena cava; d. Left renal vein; e. Left ovarian vein; f. Common iliac vein; g. Fallopian tube; h. Ligamentum teres; i. Hypogastric vein; j. Uterine vein; k. Internal pudendal vein; l. External iliac vein; m. Bulbus vestibuli. (Cranz)

thought may prove useful in elucidating hyperemesis gravidarum and eclampsia.

Another fact which supports the conception of a diversion of the blood stream during pregnancy is mentioned by Boyd (*Textbook of Pathology*). In describing the liver in eclampsia he states: 'The

necrosis is peripheral in type, being most marked around the portal vein in contradistinction to the central necrosis of acute yellow atrophy and the ordinary necrosis. *Syncytial masses from the placenta may be present in this area* " [our italics] In what other way could these syncytial masses from the uterus reach the liver except by a reversal of stream from the uterine veins into the portal system? The observation seems to the author, to whose knowledge it was brought only four years after publication of his German edition, to add strong support to his theory.

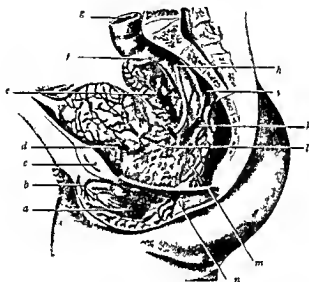


Fig. 17. Veins of the female pelvis showing anastomoses between the pudendal plexus, the vesic uterine plexus and the plexus hæmorrhoidalis. *a* Bulbus vestibuli. *b* Dorsal vein of the clitoris. *c* Symphysis pubis. *d* Middle vesical artery and vesical plexus. *e* Uterine artery. *f* Ligamentum teres. *g* Sigmoid colon. *h* Ureter. *i* Superior hæmorrhoidal veins. *k* Uterine artery. *l* Uterine veins. *m* Levator ani muscle. *n* Internal pudendal artery and vein. (Cornino.)

It is a long-established popular notion that there exists a relation between the development of varicose veins and piles in pregnancy, and the successful course of the pregnancy—a belief doubtless based upon the experiences of generations of pregnant women. Till now, however, no reasonable explanation of such a notion has been possible.

The enormous enlargement of the pudendal plexus during pregnancy is undeniable, and of this plexus the inferior hæmorrhoidal is really a part. We are not aware that any one has raised

the question of the fate of this plexus after involution. It cannot be assumed that post-partum hæmorrhoids are evidence of incomplete involution of the plexus, but if the anastomosis remains free after parturition it is obvious that any cause of portal congestion, such as an acute hepatitis, will all the more readily force portal blood into the caval system.

The peculiar phenomenon of post-partum eclampsia could be accounted for by the big drop in pressure in the pelvic veins as the uterus empties. Although the fetal source of toxins is no longer present, the normal direction of outflow of pelvic blood may be restored before the blood in the pudendal plexus—still overcharged with the accumulated toxins—has had time to pass to the liver, and if the anastomoses are still open there may be in addition a considerable reflux of poison laden blood from the portal veins themselves into the systemic circulation. It must also be remembered that during the pregnancy the liver function is in some cases impaired, so that the organ must work to the last at its maximum capacity to keep the toxins below the danger level, and thus the emptying of the uterus turns the scale, the threshold value is passed, and symptoms of eclampsia develop.

Once this argument is allowed, it may be extended to include menstruation. At this time there occur circulatory changes similar in character to, though incomparably less in degree than those of pregnancy. Several facts may be mentioned in support of this statement—the increase of all the symptoms of rectal varicose disease during menstruation, such as itching, bleeding, dragging pain in the hack, and the increased aching of varicose veins. Sometimes the pain experienced at menstruation is like that of hæmorrhoids—this suggests that during menstruation portal reflux takes place. Perhaps the menstrual congestion of the pelvic viscera is due to this. Menstrual pruritus would be easily explained by the same change. The part played by this reflux in the development of varicose veins of the lower limb will be discussed later.

The important observation that prostatectomy often results in the healing of a senile pruritus is worthy of consideration. Previously this fact has been difficult to explain, but the train of events is probably as follows. The access of the pruritogenic toxins to the general circulation through the pelvic venous plexuses is largely prevented by prostatectomy because the operation destroys and scars up a large part of the plexus. Many prostatic patients have

astonishingly large varices on the anterior wall of the rectum over the prostate. They are enormous vessels which often protrude so far through the side window of the proctoscope that care and patience are needed to replace them. Our view is, as already stated, that post-operative hæmorrhage takes place from these greatly dilated veins. 'Vein-compression' injections have just the same results as prostatectomy in that they squeeze up and obliterate these vessels in scar tissue. It is in fact more precise, quicker, surer, and achieves the desired result with less danger to life. It is to the obliteration of these anastomosing veins that the outstanding success of the method in curing pruritus is due.

INJECTION TREATMENT OF PRURITUS

During the past five years more than 680 cases of pruritus ani, vulvæ, and scroti have been treated by 'vein compression' injections. As already explained, at first only hæmorrhoids were treated in this way, but after the first hundred or so patients had been treated, the observations herein described were made, leading to the discovery of the principles underlying the method. Many of the early patients, who came solely because of rectal bleeding, reported that although the injections had caused the bleeding to disappear, an entirely new symptom, pruritus, had appeared. This itching later vanished as the treatment advanced. At the same time, other patients were volunteering that their itching and discharge, present when treatment was begun, was cured. Thus the so called obliteration treatment had in some cases produced and in others cured pruritus. These observations formed the starting point from which our routine of treatment for pruritus was developed.

In cases of pruritus, the treatment commences with 'compression' of the veins at the level of the middle hæmorrhoidal plexus. The procedure is exactly the same as for hæmorrhoids. At 5 to 8 cm above the anus a number of injections of quinine-urethane solution are placed around the bowel in a circle proceeding in a clockwise direction. Special care must be taken not to place the solution too superficially, i.e., intramucosally. The needle is made to ride over the near edge of the window and pushed on until it is felt that the point has passed the level of the far side of the window and lies in the submucosa. Until this has happened no solution may be injected. In course of time an accurate sense of the position of the needle point is acquired. If these precautions are not observed,

all three drops may be placed in the mucosa just beneath the epithelium, or the needle may pass right through the projecting mucosa, and the solution be deposited in the proctoscope. Just as with hæmorrhoids, no more than 3 times 2 to 3 drops of solution should be used at a sitting. The beginner should inject too little rather than too much. At each subsequent sitting, the injections are placed gradually nearer the anus. The nearer the anus, the more painful the injection will be, the first injections hardly being noticed by the patient. In cases of hæmorrhoids, five to eight sessions will usually be enough, but in pruritus more may be required. Nevertheless in many of the cases all symptoms will disappear after the third treatment. We have seen most severe cases in which the itching and discharge were completely relieved by the first treatment. While the patient is having injections, all ointments and pastes are forbidden. He is advised simply to keep the anal region scrupulously clean.

The larger the internal varices and hæmorrhoids, the quicker the result will be. When sphincter hypertonus and proctitis are present, treatment is more difficult and must be rather more elaborate. In cases where radiating folds of hypertrophied skin exist, with loss of pigment and hair, the varices are of the very small type and may be hard to make out on account of the œdema of the skin and the mucosa. For such cases two methods are available, both requiring preliminary local anæsthesia. Either the injection may be given without the proctoscope direct through the skin, or the instrument may be inserted and slowly withdrawn almost completely so that the region of the mucocutaneous junction can be injected through the side-window. It is a golden rule to keep the needle away from obvious vessels. Excoriations of the anal skin are not a contra-indication, they are a strong indication. After the third or fourth injection firm ridges and prominences will become palpable in the bowel wall. These are the result of the reaction of the submucous tissue to the chemical irritation of the quinine. In course of time they contract down, and by strangling the veins bring to an end the backflow of portal blood. We have never seen the vascular thrombosis described by some authors as the result of this treatment, nor is it to be expected. If quinine were prone to cause thrombosis, it would probably not be possible to give it intravenously in the treatment of malaria. The author during war service in Persia, Mesopotamia, Syria, and Palestine gave hundreds of quinine injections without ever observing thrombosis in the antecubital veins.

It is true that if some of the fluid is placed outside the vein, necrosis will often follow

As only 2 to 3 drops of solution are used at a time in the rectum, should they be accidentally injected into a vein the immediate dilution would be such as to render them harmless. It cannot be stated too often that intravenous injection is not the aim of the treatment. To inject the radiating skin folds in pruritus and the following technique is adopted. By two imaginary diagonal lines the anus is divided into four equal quadrants. With 5 cc of 1 per cent procaine solution the skin is anæsthetized, beginning the injection at the posterior commissure and infiltrating to right and left. When the area is anæsthetic, three drops of quinine-urethane solution are injected under the anal skin as the needle is slowly withdrawn. Three to four such injections may be given through separate punctures until the whole anæsthetized area has been treated. At each subsequent treatment another quadrant is anæsthetized and treated.

Since the publication of the original German edition in October, 1935, I have treated a further 180 cases of pruritus ani, making 680 cases in all. Besides these, there have been a number of recurrences. Of course, it is possible that other cases have recurred and have gone elsewhere, but about 10 per cent of the cases have returned quite readily for further treatment. Indeed, many have stated that they would willingly undergo the treatment every year if necessary, so pleased are they to have found some means of relief from their wretchedness. There has been no single case where after a successful first treatment the second treatment was a failure. The only refractory cases were those who had been treated with X rays and had never healed completely. In about 2 per cent of the most severe cases up to 20 treatments were needed, but even in such cases marked improvement was obtained. The perianal skin in these patients was as a rule markedly atrophic, and showed advanced vascular changes from previous X-ray therapy.

Other workers, both in Austria and elsewhere, who have tried the method confirm its effectiveness. For interest some typical case histories are appended. The good results seem to us strong evidence for the underlying theory, demonstrating that the varicose condition of the rectal vessels is in fact the cause of the pruritus.

Case 1—A man aged 54, had had severe itching for 18 years. He had used large quantities of ointments and suppositories in the effort to obtain relief and stated that he required an aperient daily. Seven years

previously he had received his first X-ray treatment, after which he had remained well for a year. Then the irritation returned with all its old intensity, being intolerable at night. A second course of X ray therapy produced a remission lasting 8 months, and a third gave only 6 months' relief. His fourth course had *no effect at all*.

When this patient came to us on March 22, 1934, he had an extensive eczema. The skin was depigmented, and the ordematous radial folds of skin were raw in places. After three treatments he stated that he was greatly relieved. By May 2, although he occasionally experienced slight itching, the oozing and moistness had quite disappeared. A fortnight later he was completely symptom free, and has remained so.

Case 2—An electrician, aged 34, gave the following history. In 1928 he had been seen by a certain professor of surgery, who made a diagnosis of hæmorrhoids. He was operated upon, and left hospital in fourteen days. In spite of the operation he had such severe itching and eczema he could not sleep. He next sought the advice of a skin specialist who treated him with ointments, baths and ultra-violet light. After eight weeks of this treatment, he went to another doctor, who continued to treat him a further six months without success. Next, he entered a skin clinic in Vienna, where a tar and sulphur ointment was used and finally X-ray therapy given. Two treatments arrested the itching, but after only six weeks it returned with full intensity. In 1931 he returned to the skin clinic, where on proctoscopic examination a fissure was diagnosed and operation advised. Declining operation, he left the hospital and went to see a second surgeon, who was unable to confirm the diagnosis of fissure, but recommended him to yet another dermatologist who gave him an ointment. This was successful for a time, but soon lost its efficacy. He returned to the radiotherapist, and another course of X rays gave him eight weeks' relief. After this, he tried various powders, tannin wash-outs, and a special diet. A diagnosis of gastritis was made, and the possibility of gastric ulcer discussed. At this time he began to suffer a great deal with distension, and after three more doctors had failed to help him he resorted to herbalists. These, too, were quite unable to give him relief, and on June 19, 1934, he came to me. After five treatments he was symptom-free, and the eczema had vanished. On Dec 4 1934, he returned with a small thrombosis at the anal margin, but was quite free from pruritus. After incision of the thrombosed nodule he lost his symptoms, and remains well to date.

Case 3—An office worker of 50 had suffered for a long time with burning, itching, and discharge. He was referred to me for treatment on April 15, 1934, by a radiotherapist. He had an extensive eczema with ulceration in the anal region. After six treatments he became symptom-free, and remains so to date.

Case 4—A government official of 62 came with a story of intense anal irritation for 35 years. He had been treated for this as long ago as 1898, by Professor Kaposi, without complete relief. For 18 years he had managed to make life bearable by the use of ointments and powders.

during the whole of this time there was anal eczema and discharge. On one occasion a rectal polyp had been discovered, and removed by operation, but this did not affect his symptoms. He had visited a dermatologist who treated him with locally applied ointments and later sent him to a radiotherapist. The X-ray treatment he received (1921) resulted in a painful ulcer which took six months to heal, but relieved the itching for ten years. In 1933 the trouble returned, and for a year he was treated with ointments and suppositories with little success. The eczema spread to the scrotum, thighs, legs, and extended up the back as high as the neck. Again he was under the care of a dermatologist for a year, but the eczema became progressively worse. Again he was treated by X-rays, but this time without the slightest effect. Finally the ulcerating eczema about the anus was so severe that he could not sit down, and life became a burden. He again asked for X-ray treatment, but the therapist after treating him for five days referred him to our clinic. We saw him first on March 26, 1934, when he had exceedingly severe eczema with hypertrophic granulations. After five treatments he was able to sit in comfort and was almost symptom-free. On May 4 he came again with slight itching, and after two further treatments he became completely well and remained so.

Case 5—On May 31, 1935 a 30-year old Doctor of Laws was sent to me for treatment. He had a five years' history of anal pruritus which was particularly bad at night. This had been considered the result of a gonocoeal infection and he had been given treatment by a dermatologist. As this failed he was given X-ray treatment. Then in Paris he was seen by several doctors in consultation and a diagnosis was made of prostatic enlargement, for which he was treated from September to December, 1934, with prostatic massage. At last, because the itching became intolerable and he had no peace day or night, he took refuge in alcohol and had to be sent to a psychotherapeutic clinic for treatment. He claimed that two large glasses of cognac were the only relief he could find for his pruritus. The alcoholism eventually caused him to be sent to a home. Between May 21 and June 11 he was injected with 2.5 g. of quinine-urethane solution in a series of five treatments. So successful was this that the whole anal and gluteal region, which had been one large raw surface, regained its normal appearance within ten days. The first four injections were sub-mucous, only one subcutaneous injection being needed. On June 18 he was discharged cured.

Case 6—On March 20, 1935, a police inspector of 59 was sent to us with a rash covering the whole trunk and a weeping, itching eczema of the anus. Examination showed here, as in all such cases, a proctitis, a hypertonic sphincter, and perianal skin tags. After six treatments, he was symptom free on April 18, 1935. The irritating rash on the trunk and extremities had completely disappeared after the third treatment. There has been no return of symptoms.

CHAPTER IX

ACUTE AND CHRONIC SUPPURATIVE THROMBO-
PHLEBITIS OF THE RECTUM AND ANUS
(PERIPROCTITIS AND FISTULA-IN-ANO)

By *periproctitis* is understood an inflammation of the connective tissue around the anus. Very many causes have been suggested for both this condition and fistula. A few only need be mentioned: foreign bodies in the rectum (fishbones, needles, fruit pips), direct trauma as the result of a fall or stab wound, dysentery, tuberculosis, typhoid, syphilis, prostatitis, and other inflammatory lesions of the male and female genital organs, dermoid cysts, *æcoliths*, *furunculosis*, and the downward tracking of abscesses from above.

It is possible that all these factors may at one time or another play a part, but in none of our cases were any of these things causal. The sole true cause of perianal abscess is a spontaneous thrombosis in the inferior hæmorrhoidal plexus. It is likely that in the normally healthy individual a spontaneous thrombus seldom becomes infected. In the great majority of cases, doubtless, the thrombi disappear unaided in a few weeks. All that remain are the well-known perianal skin tags which are so often treated by cauterization (Langenbeck's operation). The presence of these hypertrophic tags is invariably a sign of previous thromboses. The term 'thrombophlebitis' is in such cases of questionable accuracy, since it implies inflammation. Certainly a great many spontaneous thromboses show no signs of infection or softening.

Suppuration is a relatively rare occurrence, being found in scarcely one-tenth of the cases. When it does happen a perianal abscess results, and this condition would be much better termed 'suppurative anal thrombophlebitis'. The following types may be distinguished: (1) Marginal, (2) Intramural, (3) Pelvirectal, (4) Ischioirectal.

Consideration will show that this classification corresponds with the later course of the fistulæ which result from either the spontaneous discharge or the operative incision of perianal abscesses. The extent of such abscesses depends upon the degree of involvement of the rectal varices by the thrombosis and subsequent infection.

1 The *marginal type* is usually due to suppuration of a thrombus in the inferior hæmorrhoidal plexus

2 The *intramural type*, which commences with fevers, rigors, and severe pain on defæcation, is due to the involvement of the middle hæmorrhoidal plexus. Sometimes intense pain may be present for some two or three days before the abscess becomes obvious. Indeed, in many cases a so called internal fistula will have formed before the diagnosis has been made. It is not possible to make a valid distinction between the mode of development of a fistula and that of a peri-anal abscess.

3 *Pelvi-rectal abscesses* may, of course, result from causes such as pelvic or vertebral caries, but these must be excluded from the present discussion as they do not come within the scope of this book. They may also result from wrongly conceived extensive operations undertaken for the cure of rectal thrombophlebitis. The more radical the operative procedure, whether it be for periproctitis, for fistula, or other lesions of the rectum—or indeed of the prostate—the more frequently will these secondary abscesses be observed. Apart from this cause, such extensive spread of the inflammation occurs in only a very small percentage of cases.

4 *Ischio-rectal abscess* results from the spread of the septic thrombosis to the internal pudic plexus. The theory that it is due to tracking of pus along the lymphatics from a focus above is hard to believe. This subject will receive fuller consideration later.

Since the superior hæmorrhoidal veins on the posterior wall of the rectum have a symmetrical arrangement, it follows that thrombophlebitis when it occurs may also be symmetrical. In this case it may give rise to the not very uncommon symmetrical anal abscesses. These abscesses develop simultaneously and often lead to symmetrical fistulæ (*Fig 18*). As confirmation of the suggestion that these fistulæ are the result of a thrombophlebitis of the hæmorrhoidal veins, the fact that the tracks unite on the posterior wall of the rectum to form a main trunk should be considered. At one point in the main trunk

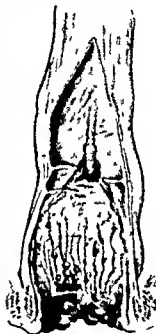


Fig 18—Symmetrical variety with two main branches

a perforation of the rectal mucosa will be found, as may be clearly demonstrated with methylene blue. *Whether of the two external openings is used, the dye will appear at the common internal orifice and may be observed with the side-window proctoscope*

A fistula originates, in the vast majority of cases, in a perianal abscess. Whether the abscess ruptures spontaneously or is incised is immaterial—a fistula may still result. If suppuration occurs in thrombosed superior hæmorrhoidal veins, the infection may spread along the clotted vessels back down the tributary veins, and down to the capillaries. After a few days, a reddened and oedematous spot will appear on the skin (or there may be bilateral foci) and softening will follow. That the resultant subcutaneous abscess is connected with the suppurative thrombophlebitis above by one or more 'tracks' (which are really venous trunks) may easily be demonstrated by injecting methylene blue. The true pathology of these lesions has until now remained unrecognized because of the peculiar anatomical structure of the region. Incision of the perianal marginal abscess only allows the escape of the pus from the lowest part of the track, that contained in the varix itself, and the higher part of the track, can only escape with difficulty unless perforation into the rectum has occurred. When the sphincter relaxes the column of fæces may help to express the pus from the varix, but after defæcation the contracted sphincter again interrupts free drainage.

The fact that peri-anal abscesses usually drain badly has led to the most diverse operative procedures. It is interesting that although the aetiological similarity of peri-anal abscess and fistula was not realized, the treatment for both lesions was identical. Surgeons formerly employed a radial incision to open the peri-anal abscess, this incision being designed to divide the sphincter at the same time. This type of operation often resulted in incontinence. Then it was stated that to spare the sphincter the incision should run tangentially to the anus from the perincum backwards towards the coccyx (Moskowitz). If our conception that peri-anal abscess is in fact nothing more than an acute suppurative thrombophlebitis is true, it puts the treatment of these so called tracking abscesses in quite a new light. Laying open the fistula, with simultaneous division of the sphincter, thus becomes exactly comparable to division of the sphincter for abscess. No doubt such treatment allows a freer escape of pus from the suppurating veins, nevertheless it should be discontinued for both theoretical and practical reasons.

At this point, attention may be drawn to an interesting analogy that as far as we know has escaped the notice of previous observers. Anal fissure, periproctitis, and fistula have been regarded as three separate, unrelated entities. It is therefore all the more remarkable that for hundreds of years surgeons have advocated the same treatment for all of them—namely, division of the sphincter. We mention this important fact, which has so far escaped the attention of workers, because there is no doubt that peri-anal abscess, fissure, and fistula can all be cured by this procedure. Surely this fact makes it probable that some very close relation exists between the three lesions.

The time has come, in the writer's opinion, to substitute an aetiological classification for the traditional anatomical one. Periproctitis should be termed *acute suppurative anal thrombophlebitis*, fissure becomes *chronic marginal thrombophlebitis*, and fistula, *chronic anorectal thrombophlebitis*. Thus the conclusion is emphasized that the pathological process is fundamentally the same, but that owing to the various stages and the anatomical variations, the three conditions have hitherto been described separately.

In 'vein-compression' therapy a method has been found equally applicable to all these different manifestations. The problem of peri-anal abscess is primarily one of prophylaxis, consisting in the obliteration of the internal varices. Since every periproctitis is preceded by thrombosis of an external, or more rarely, an internal, varix, the prophylaxis is continued by incising and emptying the clot as soon as it forms. If the clot can be removed before it becomes infected, peri-anal abscess will be avoided. The procedure must be combined with injections to compress all the veins not yet thrombosed and all possible steps must be taken to avoid portal stasis. If a thrombus is already infected, incision must still be practised. The incision should not be large and, most important of all, should be as close to the anus as possible. The farther the incision is from the anal margin, the more troublesome the resulting fistula is likely to be. Every patient who has a peri-anal abscess has certainly hæmorrhoids, because the abscess is in reality a suppurating varix. It follows that all such cases must be given 'vein-compression' treatment. A careful history in cases of peri-anal abscess will usually reveal other symptoms of rectal varicose disease.

Blond, at a meeting of the Gesellschaft der Aerzte at Vienna on June 8, 1934, suggested for the first time that the term 'fistula-in ano'

should be replaced by the more scientific term *chronic suppurative anorectal thrombophlebitis*. Gant defined an anal fistula as an ulcerous track with two orifices and without granulations, one opening is on the skin near the anus and the other is in the bowel. The weakness of this definition is at once made evident if a fistula is studied radio-



Fig. 19—Radiograph of a branching fistula after three operations

logically with the help of the quinine-iodine paste developed by the author and used for both the study and the treatment of these freely branching channels (Figs 19 20). No more satisfying than the above definition are the theories hitherto advanced to explain the origin of fistula, and treatment has never been based upon any clear understanding of the pathology.

Rotter, discussing the therapy of anal fistula, declares that surgery has not yet solved this important problem satisfactorily Tietze, of Breslau, who contributes the chapter on anorectal diseases in Kirschner-Nordmann's *Handbuch für Chirurgie*, sums up all the attempts to explain the poor healing of rectal fistulæ with the words "Why do not fistulæ heal? The answer is simple—we do not know." Gant stated quite clearly that "the ætiology and pathology of periproctitis and abscess do not differ essentially from those of fistula, because the latter is a result of the former conditions." Nevertheless, Gant accepted the common belief that peri anal abscesses are formed



Fig. 19.—Lateral view of the fistula shown in Fig. 18

by the tracking of pus downwards from above. Now though there are many allusions in the literature of the past hundred years to the idea that peri-anal abscesses owe their origin to suppurating piles, this not wholly incorrect observation has been too little heeded in more recent times, and it has never been subjected to critical examination.

What is actually meant by 'suppurating piles'? The term 'piles' refers to the varicose, dilated, anorectal veins from which bleeding occurs, situated at the anus, or higher up in the rectum. Now even though blood flows slowly in varicose veins, yet it is fluid, and if such fluid blood became infected the result would be a

septicæmia. Yet in the vast majority of cases this does not result. *In other words, the sequence must be, not infection of a varix but of a previously thrombosed varix.* Thus the term 'suppurating piles' is misleading. Better names would be *suppurating thrombosed varix*, or *acute suppurating thrombophlebitis of the hæmorrhoidal veins*.

Regarded thus, the clinical course of the lesion is easy to understand. For example, if a small thrombosed varix at the anus suppurates and discharges spontaneously, the result is an ulcer—the so called fissure, better described as *acute marginal suppurative thrombophlebitis*. If a similar thrombosed varix above the level of the sphincter should become infected, the superficial part of the vein wall necroses in the same way, and the clot separates by suppuration. Proctoscopy at this stage will reveal an 'internal blind fistula' discharging pus. In the course of a few days a larger area of vein wall is destroyed, and the opening comes to resemble a more open ulcer, which with its elevated edges is very much like those seen at the mucocutaneous junction. Such ulcers situated above the sphincter may produce almost the same symptoms as 'fissure', namely, pain made worse by defæcation. They are quite wrongly referred to as 'stercoral' ulcers. When such a high lying thrombosed varix becomes infected and suppurates, clotting is not confined to the varix but spreads along the whole vein until the entire set of vessels from the main trunk down through the branches to the capillaries is shut off from the circulation. Thrombosis may spread through the anastomosis between the superior and inferior hæmorrhoidal plexuses and reach an area of the anal skin. Thus a course is marked out which the infection may follow, and this is reflected in the symptomatology of the complaint, the patient complaining for some days before the appearance of the peri anal abscess of a sensation of heat and pain in the rectum. During this interval rectal examination is already very painful, and proctoscopy impossible without anæsthesia.

Recently the author had the opportunity of watching the progress of a case of this sort. *The patient, a man of forty came one day complaining of severe pain in the rectum.* Digital examination revealed a thrombosis high up on the posterior wall. (As these patients cannot tolerate digital examination much less proctoscopy, our procedure is to insert a well-lubricated rubber catheter into the anal canal, closing the open end with a clamp, 10 c.c. of a 1 per cent procaine solution is then injected through the catheter and

allowed to run into the rectum like a miniature clysma. After a few minutes full examination becomes possible.) In this particular case careful proctoscopy plainly disclosed the varix on the posterior wall, and two large tributaries passing upwards to enter it. Four days later he developed a high temperature. The next day a perianal abscess appeared on the left side, and within twenty-four hours of this a second abscess appeared on the right. We were in fact dealing with a typical horseshoe thrombosis which had given rise to symmetrical abscesses and ultimately to a classical horseshoe fistula (*Fig. 21*).

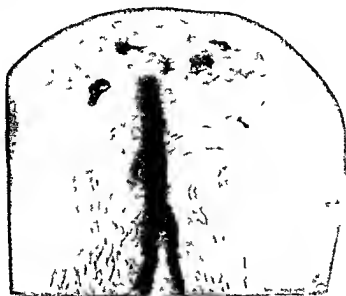


Fig. 21 Horseshoe fistula with multiple external openings

We have several times been able to demonstrate by means of the side window proctoscope the bluish black and extremely sensitive thrombosed varix before softening of the infected clot has occurred. If the illumination is good, it may sometimes be observed that the mucosa over the varix is discoloured and ulcerated, with the vein wall already showing through at one spot. Very likely it is here that infection makes an entry when defaecation occurs. These patients are generally constipated and it is easy to believe that the scybala may damage the already necrotic mucosa. The so called 'stercoral' ulcer is nothing more than the empty, open vein after the exposed part of the clot has been expelled.

The majority of surgeons believe that a peri anal abscess starts from a focus in the submucosa, and travels downwards towards the anus under the mucosa, the lymph-channels playing an important part in the spread. This erroneous conception is to blame for the nomenclature and classification in general use, which is as follows (1) Subcutaneous abscess, (2) Submucous abscess, (3) Incomplete external fistula, (4) Incomplete internal fistula, (5) Complete fistula.

This somewhat primitive classification has been brought considerably nearer the facts by an observation of Moskowicz. 'Even if there are only one or two external openings, there will often be extensive fistulous channels deeply, which may extend in a circular fashion round the rectum.' Incomplete fistulæ, according to the same writer, are in fact as a rule complete, but the internal openings may be hard to find with a probe.

These differences of opinion are easy to understand. The majority of writers have in mind only one stage at a time of the thrombophlebitis, and have never tried to relate it to any other stage. But there are several stages in the process. The acute peri anal inflammation may be seen while as yet there is no opening. It may then be converted into an 'incomplete external fistula' either by incision or by spontaneous rupture. Or an internal incomplete fistula may result if the suppurating varix bursts into the rectum. Finally, if rupture occurs in both situations, a 'complete fistula' will be the result.

Again, the stages may be observed in those cases which are healing spontaneously. If the inner end happens to heal first an incomplete external fistula is left, on the other hand if the pus drains more readily through the inner opening this may remain open longer, so that an internal incomplete stage is passed through. Moreover, a given state of affairs may become reversed during healing. A temporarily obliterated external opening may begin to discharge again, while the internal orifice in turn dries up. This may happen, for instance, if the infection flares up in some branch vessel which has not yet become obliterated. If such a vessel happens to lie nearer the outer end of the tract, the external opening will commence to discharge after an attack of local pain.

A study of the radiographs in *Figs 19 and 20* will at once reveal that prevailing ideas of the nature of fistula must be incorrect. The branching shadows at once suggest veins. The symmetrical distribution on either side of the bowel cannot be explained away as a sort

of tubular abscess made by pus 'tracking' from a submucous focus. This latter rather hazy concept has been fostered by the fallacious diagrams which are unfortunately found in all text-books. Fig 22 is an example, and the fallacy is that, although in such a sectional representation any submucous collection of pus must appear tubular, no reason is supplied why in actual three-dimensional fact the abscess should retain this shape. To put this in another way, an abscess lying in a tissue plane will tend to appear linear in cross-section. If such an abscess is pictured spatially, however, it will be seen that the diagram furnishes no reason why the pus should not spread out laterally in its submucosal tissue plane.

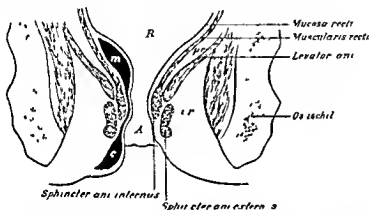


Fig 22.—Diagram (reporting) illustrate the pathology of anal fistula according to Kirschner-Nordmann. R Rectum, A Anus, c Subcutaneous abscess, m Submucous abscess, ir Ischio-rectal space, pr Pelvirectal space.

It is quite impossible to accept the current view that a submucous abscess situated above the sphincters, after spreading freely in the loose submucous connective tissue, tracks downwards as a narrow, clean-cut tube, perforates the perineal fascia in this tubular form, and then spreads widely in the subcutaneous tissue. It is still more absurd that such an abscess should suddenly, in the form of a narrow tube, track upwards or sideways in defiance of gravity. A glance at a radiograph of a fistula of this type is enough to show the untenability of these ideas. Comparison with the anatomical distribution of the veins clearly demonstrates that the symmetry of the horseshoe fistula or of the V-shaped or Y shaped fistula is only to be satisfactorily explained by accepting their venous origin. They are branching veins which have first become excluded by thrombosis from the

general circulation, and then infected. The widely held view that gravity can cause a submucous abscess to track downwards so forcibly that it pierces the perineal fascia must be abandoned. The mucous membrane under which the abscess is alleged to have been produced by trauma (from a foreign body such as a bone fragment or a fish bone) would obviously be weakened, and would certainly give way long before the intact perineal fascia, which is some distance off, and connected with the original abscess by a narrow channel only. It cannot be maintained that the fascia too is damaged by the foreign body¹. In brief, the pus would find its way much more readily into the bowel than create a long track past the sphincter and through the perineal fascia.

Similar conclusions may be reached by reasoning from known facts in this way. Thrombosis is known to occur in varices below the sphincter, and the thrombi may undoubtedly become infected. Now since the same kind of varix may be found above the sphincter, what happens when the same process of thrombosis and infection occurs in this situation? Yet nowhere in the literature has this question been clearly put, much less answered.

For periproctitis and perianal abscess it would be more accurate to substitute the term *acute suppurative anorectal thrombophlebitis*, and for fistula, *chronic suppurative anorectal thrombophlebitis*. A number of facts, previously puzzling, are now readily explained. The poor healing qualities of fistulæ, for example may be considered. Fissures and fistulæ, it should be noted, share this quality. The constant movement of the sphincter, by making rest of the ulcer impossible, has been blamed for this, and at first sight this seems a reasonable explanation, for it can be applied equally well to fistula and fissure. But if a few drops of quinine solution are injected beneath the fissure or if the latter is deepened a little with the cautery or diathermy, it will heal quite quickly, although the sphincter remains as before. It should be noted, too, that fistulæ situated at some distance from the sphincter heal even more slowly than those closely adjacent to it. Fistulæ elsewhere, as on the abdominal wall heal quite well in spite of the constant movement resulting from breathing. Another reason for the delayed healing sometimes advanced is the rigidity of the walls of the track, together with the fixity of the boundaries of the pelvic outlet in which it is situated. Though this seems plausible, the following are serious objections to its acceptance.

1 A fissure has the same bad healing powers, even though its walls may not be rigid

2 A sacral fistula, although it has rigid walls, will heal quickly if the epithelial lining is removed by excision or cauterization

3 The rigid pelvic outlet does not delay healing of an anal fistula if the track is excised, the fistula does not again become fibrous and rigid

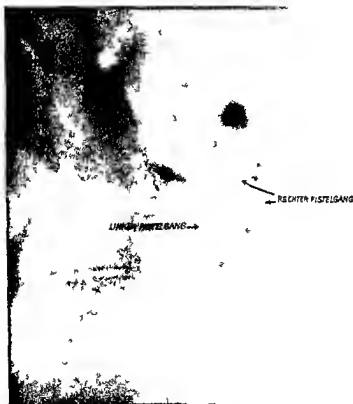


Fig. 23.—Radiograph showing rectum with two fistula tracks and a secondary branch. Arrows indicate right and left parts of tract respectively.

4 Extensive perineal wounds, as in Kraske's operation, heal quite normally, and do not become fibrous and chronic

Thus all explanations of this phenomenon previously advanced have, as Reiche and Tietze remark a trumped-up look. On the other hand, the problem ceases to exist once it is agreed that fissures and fistulae are suppurating remains of veins. They do not heal

because the infected vein wall cannot produce granulations If the fibrous wall is once destroyed whether with scalpel chemical or cautery healing follows at once The same applies to sacro coccygeal dermoid sinuses which may persist for many years with no sign of



F 24 I cl nn ruu k l k

healing but close at once if the epithelial lining is completely destroyed

Consideration of a radiograph of a fistula will demonstrate the following points (see Fig 19) Branching channels are seen exhibiting

a certain degree of symmetry. The space between them is that occupied by the bowel itself and the lipiodol has passed through the internal opening into the rectal ampulla. It will be seen that the tracks become larger in calibre as they ascend. In *Fig 19* localized dilatations or varicosities of the upper parts of the tract are clearly to be seen. If this were a gravity abscess surely the lowermost

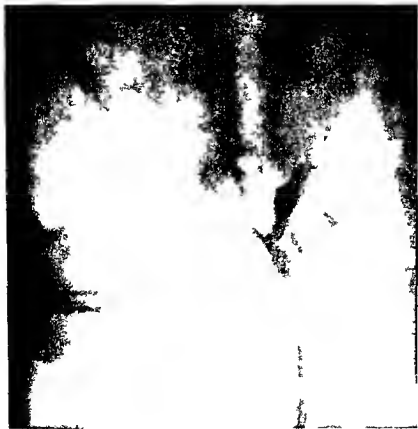
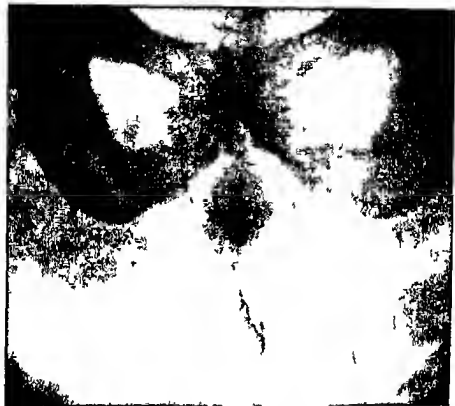


Fig 25—Y shaped fistula

part of the tract would be the widest because the weight of the pus would be the cause of the tracking of the abscess. The only structures which have the characters described—namely symmetrical distribution about the rectum, branches, and a calibre greater above than below—are the veins. The other radiographs (*Figs 23, 24, 25, 26*) are additional proof of the view that fistulous tracks are

suppurating remains of veins Study of thrombophlebitis in other parts of the body increases the probability of its truth to virtual certainty



F 26-Be d 10 f f u k

The question naturally arises why histological research has not already revealed these facts and the possible explanations are

- 1 No one as far as we know has so far considered the problem along these lines

- 2 Any remnants of inflamed vascular or perivascular tissue noted have been regarded as chance findings without particular significance

- 3 Workers have been deceived by secondary changes such as the relatively rare epithelialization of the fistulous tract

4 As histological examinations of thrombophlebitis from other parts of the body have shown, the affected vein wall may be altered beyond recognition by the suppuration

5 Benda has shown that suppuration of vein walls may cause extensive destruction of the smooth muscle and elastic tissues

6 Rokitsansky has shown that the intima and the valves disappear very early in the disease

The V-shaped and Y-shaped fistulæ which surround the rectum horseshoe fashion have as a rule only one internal opening although the external openings are frequently multiple. This is another point in favour of their venous origin, for at the anus the veins are small, numerous, and branching, but as they ascend in the rectal wall they unite into fewer and larger vessels

The poor healing qualities of a fistula are quite unlike those of an ordinary abscess, for many surgeons open an abscess in the pouch of Douglas through the rectum, yet the fistula thus artificially produced closes with astonishing rapidity. We cannot recall a single case in which an abscess so drained has resulted in a chronic fistula, although we have adopted the practice for years. Moreover, in Professor Budinger's department of the Vienna General Hospital the method has been used for very many years with excellent results, these abscesses have outstandingly good healing powers

In the great majority of cases of fistula the external opening lies close to the anus, that is, where the venous plexus is composed of very small vessels. This fact in itself is enough to suggest that the formation of the fistula may be connected with the venous network. At the external orifice there may often be seen a small raised button of granulation tissue. If pressure is applied, especially in old fistulæ, a bead of fairly thick pus may be squeezed out. These buttons of granulation tissue have often been regarded as an indication of a tuberculous lesion. Though this supposition has often been put forward and supported by more or less valid arguments, it cannot be regarded as proven. Among our own cases we have seen many typical granulation buttons in cases of fistulæ in healthy young patients, fistulæ which were certainly not tuberculous and which healed readily with injection treatment. On the other hand, among patients seen at the tuberculosis department of the Vienna Municipal Hospital, we have often seen fistulæ whose external openings had a ragged undermined edge strikingly different from that seen in healthy patients. The character of the pus in a tuberculous patient is also

different it is clearer and more watery. Nevertheless we do not believe that in these cases the fistula is primarily a tuberculous manifestation. We cannot exclude the possibility that in phthisical patients with low resistance and with tubercle bacilli present in the blood (fistulæ being as we think altered vascular structures) secondary infection with tubercle bacilli may occur and lead to alterations in the appearance of the fistula. In the same way a fistula or fissure in a syphilitic patient may take on all the characters of a syphilitic

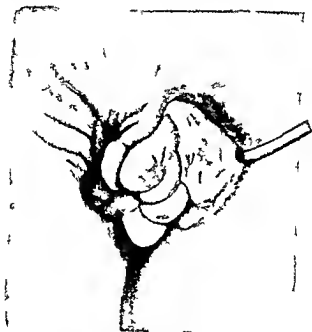


Fig. 27. Ilpe. | c skin f ds. svl. c. ce.

ulcer (Figs 27-28). It must be made clear that the outer opening of a fistula may even in a healthy individual have undermined edges.

When searching for the internal opening with a probe one sometimes has a clear impression that the point of the instrument enters a cavity. Sometimes the probe cannot be made to emerge through the internal opening even though the latter has been clearly seen proctoscopically after methylene blue injection (Figs 29-30). The reason for this is that the actual opening lies in a small tributary or a

varicose sacculæ, while the probe follows the main track of the fistula. The dye, on the other hand, which fills all the branches of the tract, easily finds the internal orifice. The custom among earlier workers was to demonstrate the opening by injecting milk into the fistula.

In the customary classification of fistulæ as subcutaneous, submucous, ischio-rectal, and pelvic, the commonest form is the subcutaneo submucous. In other words, fistulæ comparatively rarely transgress the sphincters. In our experience, the more complicated fistulæ which involved the sphincters have invariably been seen in patients who had already been submitted to operation—some as many as five times. Doubtless, too, in the production of ischio-rectal fistulæ, the wrongly conceived treatment of peri-anal abscesses has

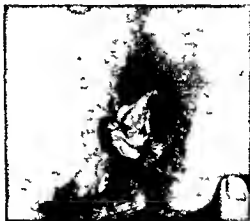


Fig 28—Syphilitic ulcer of the anus

played no small part. The tendency to make surgical treatment as radical as possible in order to avoid recurrences leads in fact to an increased number, even with simple mucocutaneous fistulæ, and a spread occurs to adjacent veins connected with the inferior hæmorrhoidal plexus. Melchior claims that 10 per cent of all fistulæ are ischio-rectal, Rotter, 20 per cent.

There can be no doubt whatever that spontaneously developed pelvic fistulæ are extremely rare. Our own experience, using the proctoscope and methylene blue for diagnosis, leads us to believe that more than 95 per cent of all spontaneously developed fistulæ are of the simple subcutaneo-submucous type, the internal orifice lying just above the sphincter, which is not involved. The natural cure of a

fistula is by perforation into the bowel the situation of this perforation should be taken as a hint that extensive operations especially those involving the sphincters are to be avoided. Even spontaneous horseshoe fistulæ with their symmetrical openings on either side of the anus in the gluteal regions do not involve the sphincters. The main channels are submucosal and reach the exterior, without penetrating the muscle by vessels running parallel to the surface.



Fig. 29.—Fistula an- sh g a s und i e external pun ng

The poor healing qualities of fistulæ have also been explained as due to constant reinfection of the track from the bowel. This view must be incorrect because —

1. Tissues exhibit the same chronicity
2. A fissure will heal rapidly if deepened with the cautery in spite of the increased possibilities of contamination
3. An incomplete external fistula whose inner opening has quite healed so that there is no question of infection from the bowel has exactly the same qualities
4. Deliberate wounds as for example after removal of polypi heal rapidly

We cannot confirm that fistulæ occur more frequently in men than in women and cannot subscribe to the view that the more supple female pelvic floor causes fistulæ to heal more readily in women.

than in men. In our experience both sexes exhibited the same chronicity if no treatment was given. Melchior discussing the clinical material at the Breslau clinic states that 60 per cent of the fistulæ were tuberculous. In this connexion it should be noted that the cases he reports had not all been personally observed by him. He makes use of material accumulated at two clinics during twenty years. The author himself admits that the records were very inadequate and that the response to the questionnaire upon which he relied was meagre.

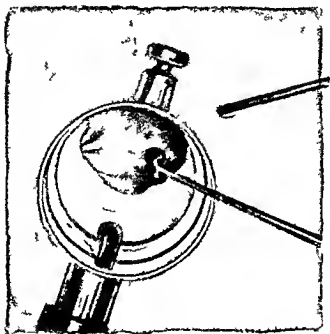


FIG. 30. External and internal opening of fistula enlarged and a probe inserted with the aid of the proctoscope.

In the last five years we ourselves have treated 200 cases of fistula and of this number less than 10 per cent raised any suspicion of tuberculosis. To study the problem of the supposed tuberculous fistula we examined 200 phthisical patients from the tuberculosis department of the Vienna Municipal Hospital and the results of the examination were compared with those from 200 outpatients at Prof. Schonbauer's clinic. Patients were chosen who came for treatment of a hernia or fracture. Of the phthisical patients we

chose only such as had positive sputum tests. There were 120 men and 80 women, and the age incidence is shown in *Table 1*.

Table 1 —AGE INCIDENCE OF CONTROL GROUP
(200 PHTHISICAL PATIENTS)

| AGE GROUP | NO. OF CASES |
|---------------|--------------|
| 20-29 years | 65 |
| 30-34 | 26 |
| 35-39 | 28 |
| 40-44 | 23 |
| 45-49 | 29 |
| 50-60 | 18 |
| Over 60 years | 11 |

The average age of the tuberculous patients was lower than that of the controls, as was to be expected from the nature of their respective diseases. Of the 200 tuberculous patients, 10 had suffered from fistula-in-ano. In 3 of these cases the fistulæ had healed spontaneously. In the 100 routine out-patient cases 3 fistulæ were found. Hence, there was no great difference in the incidence in the two groups. It must be emphasized that the phthisical patients in this series included many serious cases, some with advanced cachexia.

In our view, histological examination of a fistulous track, even if it is positive, is not absolute proof of the primary tuberculous origin of a fistula. Even less so is animal inoculation, because the patient's blood may have contained tubercle bacilli, and since the fistulæ are vascular in origin they may well have become secondarily infected.

Tavel expresses the belief that anal fistulæ develop from the so-called sinuses of Hermann. In 1878 Chiari described the mucous pouches of Morgagni's sinuses and considered that inflammation of these pouches could be the starting-point of a fistula. Fistulæ with epithelial linings, as described by Meissel and Bartholdy, are rarities. They undoubtedly occur and the epithelium is probably derived by ingrowth from the extremities of the track. The typical fistula almost without exception begins as a peri-anal abscess which has developed from a thrombosed and infected hæmorrhoid. In other words, whenever a fistula is present, hæmorrhoids must also exist. When the abscess ruptures a track is left through which feces or gas may escape.

CHAPTER X

TREATMENT OF FISTULA

ANAL fistulæ are described in the earliest medical writings. Hippocrates and Celsus recognized and treated the condition. Hume, in 1422, in his *History of England*, mentions the fact that Henry V suffered from a fistula, but that the royal physicians were unable to cure it. Jean Astruc, in a Latin dissertation written in 1728, declared that the physicians of his day knew little or nothing about the complaint.

In the seventeenth century the treatment of fistula was barbarous. Its aim was to cut out not only the fistula and its branches, but the rectal wall, and to cauterize it with a hot iron or corrosive chemicals. Most of the patients died of pyæmia, and those who survived were left with incurable incontinence or stenosis. Hence the operation was greatly feared and physicians devised all sorts of other treatments in order to avoid it. Esmarch has written about the illness of Louis XIV, telling us that the king's terror before his operation was inspired by the bad results he had seen in others. It was only after he had had all known remedies tried out on those of his subjects who suffered from fistula without any success that in 1686 he finally agreed to submit to operation for his own relatively simple fistula. For that operation his first surgeon, Felix, specially invented a combined probe and knife. Afterwards many of the king's subjects were operated upon with this same 'royal bistoury.' The operation consisted in simply splitting open the fistula.

In 1887 Greffrath wrote a detailed review of the operative treatment of fistula. The cautery, he says, was well known and used in ancient times. Interestingly enough, Celsus advised the injection of irritants if the fistula was too extensive for surgery. During the Middle Ages caustics were very popular. These remedies were applied by means of a knotted string. Vigo, as an adjunct to the treatment, used a laminaria dilator. Dionis, who also has left a description of Louis XIV's illness, states that in Paris physicians used to destroy the fistula gradually with bluestone, starting at the external orifice, in such a way as to convert the fistula into an open

wound Herzberg at that time used a corrosive sublimate paste, while Dzondi recommended caustic alkali. Copper sulphate, and nitric acid were employed by Fabricius of Aquapendente, by Marchetti, and by Fallopiæ. Albukasim introduced the hot iron in the therapy of fistula. Bonnet, of Paris, reported in 1853 good results from the use of tincture of iodine as an obliterating agent. Cooper reported two successful cases in which he had obliterated the fistulous track with a mixture of corrosive sublimate, limewater, and port wine.

In 1844 A. Mayer devised the technique of subcutaneous division of both external and internal sphincters, the object of which was by abolishing the sphincter action completely to prevent the accumulation of pus and fæces at the inner opening of the fistula.

Excision and extirpation of the fistula were described and practised even in the time of Celsus. Excision was carried out by passing a flattened and grooved probe along the fistula into the rectum, the point being then brought out at the anus. The tissue between the fistula and the bowel was thus put on the stretch, and excised by making two parallel cuts on to the probe. French surgeons had a way of excising the fistula with a pair of polyp-shears. The fistula was seized between the two blades of the shears and excised. Hugo of Lucca, Fallopiæ, and Petit all used a method of partial excision. Laying open the fistulous track was a method praised by Hippocrates and Galen and their pupils because of its relative simplicity and quickness. For the purpose Galen used a special instrument, the *syringotome*, which was a pointed and curved knife. The royal bistoury with which Felix operated upon Louis XIV was really a modification of Galen's *syringotome*. This royal bistoury had at its tip a flexible probe-point. During the years which followed, this instrument was much modified and elaborated, especially by Garengnot, Drummond, Brambilla, and Savigny, all the improvements being based on the '*gorgeret*' originally introduced in 1664 by Marchetti. This consisted of a large grooved sound with a handle set at an angle, and fashioned of wood—according to Dessault's description—or of horn so that it would not blunt the point of a knife. It was passed into the rectum and received the tip of a small grooved sound passed along the fistula. Then a knife was slid along the fistula and made to cut against the large sound, thus dividing the tissue between.

"All these complicated instruments were discarded in 1765 by Pott. He operated with his slightly curved bistoury, with rounded

or bulbous tip, an instrument which is still in use to-day. When using this, he introduced a finger into the bowel as a guide. Voltolini, again, advocated the use of scissors, putting one blade into the fistula and the other into the rectum. The track was then split open by closing the scissors. After Pott had shown that to ensure healing it was unnecessary to destroy all the scar tissue, simple laying open alone sufficing, operative treatment became more generally adopted by the profession." (Quoted from Greffrath.)

The ligature method, known in Hippocrates' time, was re-introduced in the Middle Ages by Dessault. A lubricated probe was passed through the fistula and the tip grasped by a finger introduced into the rectum. The probe was now used to draw a silk thread through the fistula. The ends of the thread were tied tightly enough to compress the tissues it embraced. After two or three days, the loop which had become slack was again tightened, and this was continued until the increasing granulation and scarring healed the fistula. Later, linen, hemp, and horsehair were tried instead of silk, as well as wires of lead, tin, silver, and gold. Where the internal opening was high up, a modification was to push up through the fistula a strand of catgut, so that its end passed into the bowel, and then to coax this end out at the anus by means of enemata. Later on, various instruments were designed to reach and pull down the end of the catgut.

In 1856, the French surgeon Chassaignac evolved a bloodless method of dividing the sphincter, using a crushing clamp or 'ecraseur'. In principle this instrument was not unlike the enterotome which is now used to destroy a colostomy spur. In Esmarch's opinion "the introduction of the crushing instrument into the fistula was very troublesome and difficult."

The galvanocautery introduced to surgery by Mitteldorpf offers another method of bloodless operation. A loop of platinum wire is attached to a probe and guided through the fistula into the bowel, the end being brought out at the anus. The two ends are now connected to a special holder, and by means of a battery a current is passed along the wire. This becomes red-hot, and lays open the track. Though the technique is practically bloodless, it has the disadvantage of requiring complicated apparatus. Esmarch (in *Pitha and Billroth*, 2a, 125) highly recommends the method, as shown in the following extract: "In those cases where it is wished to avoid loss of blood, the galvanocautery method is an excellent substitute for operation."

The first man to take social considerations into account when selecting his treatment appears to have been Strohmayer. In his *Handbuch der Chirurgie* (vol 2), he writes "In dealing with a fistula, the choice lies between surgery and the ligature. Throughout treatment by the latter method, the patient can follow his employment, which more than compensates for the somewhat longer time taken."

Esmarch's views are "The simplest, quickest, and most certain method of curing fistula-in-ano is by incision. Where this bolder plan is ruled out by anæmia or debility due to previous or coexisting illness, the use of the ligature is the only worth-while method."

Most of these methods of treatment have now passed with the instruments into oblivion. The practice of obliterating the fistula and the chemicals suited to the purpose, so well understood by the ancients, have been totally neglected by present-day surgeons. For this, the teaching that surgical treatment is always satisfactory is largely responsible. Thus Strohmayer, writing upon the question in his *Handbuch der Chirurgie* (1851) teaches "Injections into the fistula and cauterization should no longer be used." He had he says seen serious misadventures result from these methods. Nevertheless, the unprejudiced must admit that no misadventures could be worse than those which were apt to follow the often barbarous procedures of the older surgeons, with their terrifying mortality. All the same there must have been certain definite reasons why the obliteration treatment was put aside and forgotten and among them were probably the considerations that success or failure of the treatment depends upon —

1 The choice of chemical. Thus zinc chloride is useless but quinine may be used successfully.

2 Its dosage. (Though $\frac{1}{6}$ gr of morphia is a useful remedy, an overdose may prove fatal.)

3 The manner and site of its application, just as curari though a poison if given intravenously, may safely be given per rectum.

It was simply because these facts were not fully appreciated by older workers that the obliteration treatment fell into discredit. We might illustrate this by an analogy with the early days of X-ray therapy. If the early workers had had the modern powerful apparatus at their disposal, their primitive experimental attempts might have been so disastrous as to lead to the abandonment of the method. Fortunately, the pioneers of radiology had such relatively poor tubes

that even though in some cases they treated patients for hours at a time their results were tolerable enough to warrant continued effort

In the case of the present author it was the bad results of surgery which had the effect of turning his attention to a simpler method. There are, Reichle and Tietze claim, fistulæ so extensive that a cure can only be effected by extirpation of the rectum, but such cases are probably not spontaneous fistulæ but the result of ill-conceived operations

The two most important operative methods nowadays are (1) Splitting open the fistulous track, and (2) Extirpation of the track. The former is performed under local anæsthesia. The sphincters are stretched and the track laid open along a probe. The wound is cleansed, bleeding stopped, and the cavity plugged. The patient is placed on a fluid diet and given opium for two or three days. An aperient is then given. The plugging may need to be changed under general anæsthesia. Incomplete external fistulæ are converted into complete ones by making the point of the probe penetrate the bowel wall. Now though this treatment in the simple submucous type of fistula may be carried out without damaging the sphincters, and though it is in general relatively free from danger, yet in those cases where the inner opening lies high above the sphincters the procedure may become not only difficult but dangerous. We have seen, too, complete incontinence result, and sometimes recovery has been very protracted and complicated by terrible infection almost fatal in its severity. Smith and Lange of New York described in 1886 a combined splitting open and excision technique with suture. Their method was adopted and modified by Sendler, Rotter, König and Von Hacker. We consider that the method of König and Von Hacker is only applicable to rare cases because (1) It cannot be used where there are a number of branching channels. (2) The internal opening is situated in a suppurating varix and stitches here are prone to cut out, (3) It is impossible to avoid tearing older and tougher fistulæ which results in incomplete removal. How far surgery falls short may be gathered from a consideration of the elaborate methods of Moskowitz. He opened up the fistulous channels widely and endeavoured to cover the raw areas with skin flaps, but in spite of this recurrences still took place. We have seen cases in which there were more external openings after this operation than before. It would seem that the more ambitious the intervention, the less chance there is of quick and sound healing.

INJECTION TREATMENT

During the last four to five years we have treated 200 cases of fistula with injections, and we consider this treatment much more sound in principle than operation. We do not imply that in favourable circumstances a simple incision may not be the quickest method, but no definite rule can be laid down, the choice of method being largely a matter of experience. The technique is based on the following facts —

1. *Fistula in-ano* is a suppurative thrombophlebitis and usually affects a group of anastomosing veins. These veins by previous thrombosis have been shut off from the circulation.

2. The underlying cause of the fistula is the hæmorrhoids.

It follows that the treatment of fistula must begin with the treatment of the hæmorrhoids, for as has been stated there are always high varices in these cases. If a beginning is made with the 'vein compression' of the varices of the middle and inferior hæmorrhoidal areas, it will be found that many fistulæ, even chronic ones, will dry up without further treatment. Often three to four sittings will suffice. *Fistulæ* which have never been interfered with by surgery, but are entirely spontaneous, respond best of all. *Fistulæ* with free discharge often show striking improvement, especially if the varix in which the fistula originates is becoming compressed. The fistula may become quite dry. As a rule, however the fistula does not close spontaneously even after the course of injections has been completed. In those cases which have been operated upon (i.e., recurrences) it is essential to make out accurately the extent, course, and length of the main track and its branches. The most resistant type is the recurrent external incomplete fistula with rigid walls.

The examination of a case is conducted as follows. The hyper-tonus or hypertrophy of the sphincter may make the examination difficult and as no unnecessary pain should be caused, it is best not to attempt too much at the first sitting. With sensitive patients it is advisable to run into the rectum 10 c.c. of a 1 per cent procaine solution before proctoscopy. However, even before endoscopic examination, the position of the internal opening may sometimes be ascertained in one of the following ways. If the lubricated forefinger is passed into the rectum, it may discover the small indurated area or the little depression indicating the site of the inner opening. The opening should not be sought above the *pars sphincterica*, it usually lies not more than two to three centimetres from the anus. If the

opening has been thus located, a soft, flexible, olive-ended probe may be introduced through the external opening and readily guided into the bowel. Where these manœuvres are not successful, an injection of methylene blue (1 c c) or hydrogen peroxide may be used to show up the inner opening. The injection is given with a needle, the point of which has been removed in order to avoid transgressing the wall of the fistula. After the injection, all the varices connected with the fistula will be seen through the proctoscope bright blue against the pink mucosa. On rotating the instrument the blue dye will be clearly seen flowing from the inner opening. Even where there are several outer openings, the internal one is usually single and is connected with all of them. The solution will reach the common internal opening if injected into any of the outer ones.

A clearer idea of the course of the fistula may be obtained by a radiograph. For this purpose we make use of a paste, put up in tubes, containing lipiodol and 20 per cent quinine. At body temperature this slowly melts and enters all the fistulous tracks. The paste serves a double purpose—it gives a good X-ray picture and at the same time starts the obliteration of the fistula.

The extent of the fistula having been satisfactorily determined, the treatment may be commenced. After testing the patient for idiosyncrasy to quinine, 0.5 c c of quinine solution may be injected into the fistula. For this the non-pointed needle is again used. Not more than two injections should be given during the first week, and after this the patient should be treated only once weekly. At each sitting the shrinkage of the internal varices can be observed. As a rule it is best to give the submucosal injections before beginning the treatment of the actual fistula. Even after the third treatment in many cases the pain will go and the patients feel much improved. One patient who had had a chronic fistula for years, with so much discharge that he had difficulty in keeping himself clean, put on 15 lb in weight after the first few treatments. In resistant cases, and where the discharge is scanty but thick, it is helpful after three or four treatments to curette the channel with a long, sharp, flexible spoon. While this procedure would be very painful if not impossible during the first stages, it is well tolerated later on and produces no discomfort.

After the first treatment, fistulae behave in the same way as fissures. It will be remembered that for the treatment of fissures a local anæsthetic is necessary for the first injection, while

subsequently the fissure becomes almost completely insensitive. This applies also to fistulæ. It may be desirable to prolong the action of the injection on the walls of the fistula, and the solution may be prevented from escaping at once by placing a small pad on the external opening while the internal is controlled by pressing with the left forefinger in the rectum. Where the fistula is incomplete the pad may be retained in place with adhesive tape. Nevertheless the solution can act only for a very short time on the fistula walls, and for this reason we have adopted the quinine paste in difficult cases. We have had constructed for the purpose a fistula syringe similar to those formerly used by dentists in the treatment of dental sinuses (*Fig 31*). The piston is removed and the syringe filled with the paste, the piston is then replaced. The syringe has a short, conical, screw-on nozzle which when introduced into the fistula,

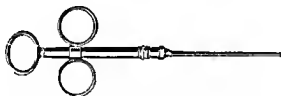


Fig 31 —Blond's paste syringe for the treatment of fistula

entirely blocks the opening. The nozzles are available in various sizes.

When treating bad fistulæ with extensive arborizing channels both doctor and patient must have plenty of patience. Usually one treatment a week is sufficient. Sometimes it happens that the external opening closes very rapidly, before the side-tracks have become clean and healed. In such cases after apparent closure, there may be sudden onset of pain in the region of the fistula which reopens and begins to discharge again. If the track is explored again with a probe it may now be found to run in a direction different from the original. This is because the previously patent track has become obliterated while another branch has opened up. For example, the fistula may be Y-shaped and one limb may be obliterated by treatment before the other. If the internal opening was by way of the obliterated limb, the result is now an incomplete fistula the remaining limb ending blindly in the soft tissues. In general, however, once a fistula begins to heal, it will close permanently. Nor

is it necessarily an unfavourable sign if a fistula temporarily reopens during treatment

Although the results of this conservative treatment are excellent, there are certain cases where a cure is more rapidly obtained by a simple laying open of the fistula. Short tracks, especially those at the posterior commissure, which are partly epithelialized either at one end or the other, do not call for obliteration therapy. The best treatment is as follows. They generally open below the sphincter, 1 to 1½ cm from the anal margin, and they may be treated by injecting a few drops around the track, or after anæsthetizing the area, a diathermy needle may be introduced into the channel to coagulate its walls. The diathermy treatment is followed by injection of quinine paste. The quickest results, however, are obtained by laying open the fistula with the diathermy needle. This simple, bloodless procedure can be performed in the out-patient department. The left forefinger is introduced into the rectum and the point of a flexible sound passed through the track and brought out at the anus. With the sound in place, a 1 per cent procaine solution is injected around the fistula. No more than 15 c.c. need be used. The soft tissue stretched upon the probe is then divided with the diathermy needle. This may be done without any loss of blood. The track is completely destroyed, the probe conducting the current and serving to coagulate the posterior part of the wall. Most of our patients were able to return to work on the day after such an operation. The result is a smooth wound which is epithelialized in about a fortnight. The treatment must always be followed up by proctoscopy, and all internal varices must be treated.

We are strongly opposed to operating on old, deep, recurrent fistulæ—the results are uniformly bad. A few case histories of such patients who had undergone these operations, some by the most eminent surgeons, will serve to illustrate this.

CASE HISTORIES

Case 1—Mr St was suddenly taken ill in Istanbul in October, 1933, with a perianal abscess. This was incised by the surgeon at the Jewish Hospital, and before the operation examination of the urine revealed a slight trace of sugar. At first, the post-operative course was uneventful. On the eighth or ninth day, when he had been given several aperients and an enema because he was constipated, he suddenly developed severe abdominal pain with collapse and paralytic distension. Twenty-four hours later he had high fever and showed all the signs of a severe

extraperitoneal pelvic cellulitis. So rapidly did the condition progress that incision was considered useless. His condition was already so bad that the extensive incisions needed would have been very dangerous. During the next thirty six hours the cellulitis reached as high as the kidney on the right side and then ceased to extend. Unfortunately the ileus persisted and in spite of the lessening of the inflammation he became steadily worse. In the hope of relieving him an ileostomy was attempted but when the abdomen was opened under local anaesthesia examination showed coils of



Fig. 3. Showing eight fistula openings after three operations. All the fistulae perianal perianal scars.

small bowel matted together with fibrin in the right lower abdomen. The operator feared that he would open into a collection of pus and thus precipitate a general peritonitis so the abdomen was closed without any further interference. The following night the patient by now moribund began spontaneously to expel flatus. Next day he was better and he gradually localized an abscess in the right side of the pelvis. This was incised and evidently communicated with the retroperitoneal cellular tissue. The patient now made steady progress but three weeks later developed a left sided abscess which fortunately opened spontaneously through the original rectal incision. There still remained when he left hospital an extensive induration of the extraperitoneal tissues on the right side and a

moderately large internal fistula from which pus escaped. No reason was ever discovered for his misadventures. After his recovery the glycosuria disappeared. The patient came to Vienna on January 3, 1934 bringing with him notes of these happenings. The abscess in the right lower abdomen had then recrudesced. From January to May he was given short wave diathermy treatment. On May 15 his fistula was again operated on and he was in hospital for three weeks. On January 11, 1935 he came under my care. Examination revealed an extensive fistula about eight inches long and very large internal piles. The latter were first dealt with in a few

treatments and during this time the secretion from the fistula previously unusually free became progressively less. By the middle of March he had received thirteen injections each of 0.5 cc of quinine urethane solution into the fistula and the internal varices and he was so much improved that he was able to undertake a three months business tour.

He noticed furthermore that the colicky pains of biliary origin from which he suffered had become less frequent. At the beginning of June he returned to Vienna and resumed treatment. After four more injections of quinine paste the fistula finally healed on July 3.



F 33—The same patient as in Fig. 32 after two months treatment.

Case 2—Figs. 32 and 33 are of a man of 42 who had been operated upon in 1924 for a perianal abscess. The incision never completely healed but developed into a fistula. Though he was sometimes free from pain there were periods of severe discomfort in the region of the fistula. In 1929 he submitted to a second operation. After temporary improvement his condition again deteriorated markedly. He began to get severe

pain on defæcation, became constipated, and his general condition suffered. There was also bleeding on defæcation. The residual fistula discharged very freely. In 1932 his appendix was removed. When he came to me on October 18, 1934, there was a typical horseshoe fistula with several openings on either side of the anus. He has remained under our care since then and has gained 15 lb in weight. Secretion from the fistula is minimal, and he is able to do full work. The fistula is entirely closed except for two small external openings.



Fig. 34—Rectal prolapse after operation for fistula

Case 3—Fig. 34 is from the case of a dressmaker of 37. She was first operated on for fistula in 1908. The operation was not successful and the fistula continued to discharge for ten years until in 1918 she submitted to a second operation (plastic repair). In 1931 there was a third operation, in which the track after being split open was treated with the Paquelin cautery. Still it refused to heal.

On September 29, 1932, she was sent to a clinic where after examination an 'anal papilloma' was diagnosed. This tumour was freely excised, and the histological findings were 'Papillary tumour. The covering epithelium is in parts stratified squamous with keratinization. There is

generalized chronic inflammatory infiltration of the stroma, in places the irregularly arranged cells with abnormal nuclei and many mitoses suggest early carcinomatous change" This is therefore an example of carcinoma developing in a chronic fistula

Case 4—L W, a man of 50, had suffered since 1910 from rectal troubles—itching and burning, constipation, and bleeding at stool In 1911 he was operated on for a fistula in ano, and for a few years he had very little trouble In 1932 his symptoms again became very severe, and in that year a second operation and in the following year a third both failed to relieve him Latterly he had developed very bad pain on defecation There was blood in the stools On June 28, 1934, he came to me with a

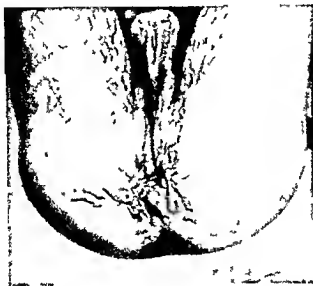


Fig 35—Anal region after three fistula operations in a long standing case showing numerous fistulous openings

suggested diagnosis of anal carcinoma arising in the old scar The suspected area was excised, and the histological report was negative There were four fistulous openings, numerous scabs around the anus, and a fissure near the anterior commissure The fistula discharged thick creamy pus (*Fig 35*)

After a few months of treatment the patient's condition was much improved (*Fig 36*) and he was able to resume work after years of enforced idleness

Of 200 cases of fistula treated by us 150 are considered healed, 20 greatly improved with complete relief of symptoms, and the remainder were either still under treatment or had failed to complete their course A few notes of typical cases are appended —

A woman of 52 whose last operation had been in 1918 was found to have at her first examination 8 fistulous openings (May 3 1934) We treated her until June 30 1934 Follow up examination in November showed all the openings healed

E T a man of 37, came to us on June 2 1934 He had been operated on eleven years before for a fistula which was declared to have been tuberculous It continued to suppurate throughout the eleven years After ten treatments he was discharged as cured on October 4 1934



Fig 36—The same case as in Fig 35 after a few months' treatment. Many of the tracks have already healed up.

S F a man of 40 has pulmonary tuberculosis Six years previously he was operated on for a perianal abscess and he had had a fistula ever since The fistula had been unsuccessfully treated four years later with cautery and diathermy He came to me on August 9 1934 and on November 22nd after thirteen treatments the fistula was closed The following month it reopened for a time and the patient was given further treatment Several injections of the solution and the paste were given

and by May 29 1935 the fistula was perfectly healed From that time he had no rectal symptoms and gained many pounds in weight The sputum still contained tubercle bacilli

A man of 54 gave a history of a fistula resulting from a perianal abscess in 1927 The fistula had been treated with caustics but without success On October 31 1934 he came to me for treatment of a complete fistula He was discharged cured on March 18 1935 after sixteen treatments (Fig 37)

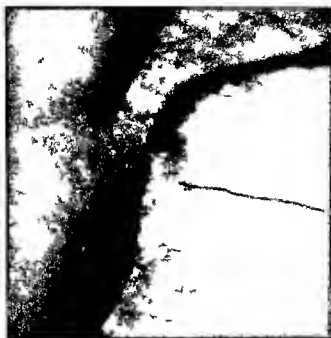


Fig 37—Show a fistula healed by injection treatment almost without a scar

L I a man of 60 came on November 22 1934 with a diagnosis of fistula The track was about ten inches long Treatment continued until June 1935 there being thirty eight sessions in all After this the long callous fistula was completely obliterated and the patient was discharged on June 19 1935

M M a man of 69 came on May 4 1934 with a fistula of six years duration On May 9 after three treatments the fistula was closed

H P a woman of 48 came on April 24 1935 with a fistula that had been operated upon three years previously On May 29 1935 the fistula was healed

S S a woman of 40 with tubercle bacilli in the sputum came on June 28 1934 for treatment of a fistula This was cured in twenty seven sessions

A doctor brought his wife, aged 40, to us, to be treated for a fistula. This had been operated upon ten years before. Whenever the fistula was closed she suffered from typical biliary colic. These attacks were always relieved when the fistula reopened and drained. She came under our care on June 7, 1934, and on January 22, 1935, she was healed. A few months later a small new suppurating track appeared, but soon yielded to treatment. By April 19 she was discharged cured.

Dr. H. F., a lawyer of 45, had been operated upon in 1917 for a perianal abscess. Since then he had suffered from a fistula. He came to us on April 4, 1935, and after five treatments was discharged on May 16, 1935, with his fistula healed.

H. N., the 66 year old father of a doctor, wrote to his son telling him that he was to be operated upon abroad for a fistula. The son brought him to us in Vienna. He came on October 2, 1934, and after fourteen treatments was able to leave Vienna on November 3, symptom free and healed.

A. H., a man of 60, came on October 8, 1934, with a fistula for which he had had an operation twelve years previously. He was discharged cured after eleven treatments on February 11, 1935.

H. D., a man of 32, came on August 2, 1934, with a fistula of six months' duration. It was healed on October 29 after twelve treatments.

B. J., a woman of 44, attended on October 31, 1934. She had first been operated upon for fistula in 1926, and since then had had several unsuccessful operations. She gave a story of attacks of biliary pain which always coincided with a lessening of the fistulous discharge. She remained under my supervision for several years. Her symptoms after treatment became very slight indeed, and the fistula, which was originally about twelve inches long, would remain dry for weeks together. At these times there were no biliary attacks. The fistula reopened several times after apparent healing, on one occasion it was deliberately reopened with a probe because the gall bladder pain recurred. A thin yellowish secretion drained away. Although this patient was never completely cured, she was well content with the result and gladly attended from time to time for further treatment.

A. B., a woman of 43, was first operated upon in 1926 and since then has had four further operations. She came to us on June 8, 1934. There were several fistulous openings. All these, except one short track, closed with treatment. Secretion became minimal, whereas it had before been purulent and copious. During the course of her treatment, a number of indurated cords previously palpable, disappeared. Eventually there remained one soft, external opening, which would scarcely admit the end of a probe. At this stage the patient unfortunately ceased to attend.

M. N., a woman of 29, had submitted to a fistula operation seven years before. In 1932 the fistula reopened and discharged freely. She was first seen by me on May 8, 1934, and was discharged cured after thirty treatments in February, 1935. During the treatments the fistula closed several times, but had to be reopened because of local pain.

Only brief extracts of the notes have been given, but they enable the reader to distinguish three types of fistula patients (1) Those who have never been operated upon for the fistula, which developed spontaneously from a perianal abscess that either ruptured or was incised, (2) those who had already had one operation, and (3) those who had had several operations. These last are the most resistant because tough areas of scar tissue are present, which render the obliteration of the vascular remnants difficult.

Patients who after a period of months came back with a recurrence, seeking further treatment, were exclusively those who had had operative treatment before coming for injections. The study of recurrences is always of particular interest, because from these cases the defects and dangers of a method are learned. In this instance two facts have become obvious (1) Almost without exception, the external openings in these recurrent fistulae are situated at a distance from the anus, and (2) Spontaneous fistula arising in a peri-anal abscess is the easiest to cure. These two observations have driven me to the conclusion that it is some mistake in operative procedure (of the fistula or the preceding abscess) which is primarily responsible for the failure of the fistula to heal. A 'false route' in opening the abscess seems to be the chief error. The author himself has now developed a technique based on this view, and no longer sees any fistulae after opening peri-anal abscesses. Since the abscess is the potential first stage of a fistula, it is to be treated as such. It must be incised as near to the anus as possible, and a grooved sound passed in, at the point where the index finger in the rectum can most clearly feel the point of the sound the latter is thrust through the mucosa, and the intervening tissue cut through in exactly the same way as with fistula. I have already mentioned that the course is in the great majority of cases superficial to the sphincter. The muscle is therefore not damaged by this procedure, and the highest point at which the sound is likely to perforate the mucosa is only 1 to 2 cm. above the anus.

Of the 25 per cent of fistulae which were not completely healed by the author's method, about a fifth failed to come back for further treatment. The state of the others was so greatly improved that they preferred to remain under my care. In many, closure of the fistula for a time (sometimes several months) was secured, it then reopened—perhaps in a new place—and began to discharge a scanty secretion. Yet not one of these patients asked to have an operation.

they were as a whole well content with what had been achieved. Sometimes even in these cases surprising successes have been attained by curetting the fistula with a small sharp spoon, filling the fistula with quinine paste, and treating the internal varices. Recto-vaginal fistulæ have been found most resistant, and hardly to be influenced at all by our treatment. This may be attributable to their shortness and to their complete epithelial lining.

These patients all tend to put on weight, and to lose the pale, subicteric tinge so often present. A certain number who, like some of the examples just quoted, suffered from biliary symptoms volunteered the information that these troubles had cleared up with the treatment. For many years the author has concerned himself with the study of gall-bladder function (*see Bibliography*, p. 134), and the yellowish colour of the sclera and the frequency of biliary symptoms in fistula patients has impressed him for a long time. It is impossible in the space of this book to go further into this subject, and the reader who is interested is referred to the papers written by the author on the gall-bladder, in which current ideas about the functions and mode of emptying of the viscus are subjected to critical study. The bearing of the matter on the subject of fistula is the close relation between the disturbances of bowel function and the biliary apparatus. The same yellowish tinge mentioned as occurring in fistula patients may be observed also in gastro-enterostomy patients whose stoma is not working well, and in those with gastro-jejunal ulcers. The author, in a series of papers, has endeavoured to show that so-called 'vicious circle' after gastro-jejunostomy, acute dilatation of the stomach, jejunal ulcer, duodenal ileus, and progressive invagination of the jejunal loop through an anastomosis into the stomach, are functional disturbances of spastic nature, to which portal congestion leads the way.

Increased knowledge of the functions of the portal circulation will certainly lead to a much better understanding of these and many other pathological conditions of the stomach, gall-bladder, bowel and rectum. A gastric or duodenal ulcer, an acute cholecystitis, a rectal ulcer (fissure), and a rectal thrombophlebitis (fistula) may all be close relations, the purely morphological names given to which have hitherto led us to think of them separately. The far-reaching effects of portal congestion or stenosis are still imperfectly realized and remain a wide field for future research. In the following chapter only one of the many aspects of this question will be considered.

CHAPTER VI

THE ROLE OF THROMBOPHLEBITIS OF THE
HÆMORRHOIDAL VEINS IN THE AETIOLOGY
OF BILIARY DISEASE

If it is accepted that anal fissure, anal fistula, and perianal abscess are all results of thrombophlebitis and infection occurring at the junction of the portal and the caval venous systems, there naturally calls for consideration the question of what effect this infection may have upon the organs to which the portal blood flows, namely, the liver and the biliary tract.

The following speculations have been stimulated in the author's mind by his observation of some 200 cases of fistula and several hundred cases of fissure. It has not infrequently happened that a fistula during the course of treatment by sclerosing liquids and pastes will close for a time, only to reopen and drain again. Occasionally such a closure (and this applies also to spontaneous closure) is the signal for a typical attack of what appears to be biliary colic. These pains recur at intervals as long as the fistula remains closed, and disappear when it reopens. Another observation is that patients who have been subject to biliary symptoms have lost these as the fistula gradually healed under treatment. These apparent coincidences were noted often enough to lead the author to suspect some interrelation between the two complaints.

It has been pointed out that closure of the external opening of a fistula may be accompanied by shivering attacks. This suggests some synchronous invasion of the portal system by bacteria.

Now there is a good deal of evidence to show that biliary disease is intimately connected with infections of various kinds. Thus it is well established that the causal organisms of typhoid and paratyphoid are excreted by the liver through the biliary tract. Posselt has pointed out that gall bladder disease not infrequently follows sooner or later after an attack of paratyphoid fever. In dysentery and cholera also the causal organisms may settle in the gall bladder. Cholecystitis, too, is often preceded by a hepatitis. Appendicitis has often been suggested as a possible aetiological factor in gall-bladder

disease, thus raising the question whether infection reaches the liver by the lymphatics or the blood-stream. The association between gastro-intestinal ulceration and biliary disease is also notable.

It has often been debated, too, whether infection reaches the biliary system from the liver in the bile or by a lymphatic spread. The following generalization is put forward in the endeavour to simplify these various problems: *The liver may become infected from any lesion of the gastro-intestinal tract, the intact mucosa is the barrier which normally prevents the ingress of infection.* In diseases such as typhoid, paratyphoid, dysentery, cholera, and gastric or duodenal ulcer, the breach in the mucosa forms a port of entry for pathogenic organisms into the portal system.

Turning now to the particular case of the rectum and anal canal, we have to consider how far suppurative diseases of the rectum such as infected thrombophlebitis at the caval-portal junction, and even piles themselves—for since these bleed they must constitute breaches in the mucosa—may allow access of infection to the portal tract. In order to obtain some evidence on these points 100 cholecystectomy cases from Professor Schonbauer's surgical clinic were asked to present themselves for follow up examination. At the same time, 100 other patients who had come for minor troubles such as fractures, and were presumably healthy were chosen (Control Group I). In this group there were 52 men and 48 women. A further 100 cases (Control Group II) were selected from patients whose histories gave no hint of rectal varicose troubles. The age incidence in each group is set out in *Table 2*. The patients in all the groups were examined with the proctoscope, and in all groups the histories were carefully retaken with regard to previous appendectomy, gastric or duodenal ulcer, typhoid or dysenteric infection, constipation, and the appearance of blood in the stools. The statistics

Table 2—SHOWING INCIDENCE OF SUPPURATIVE RECTAL DISORDERS AMONGST CHOLECYSTECTOMY PATIENTS AND TWO CONTROL GROUPS

| AGE | CHOLECYSTECTOMY PATIENTS | CONTROL GROUP I | CONTROL GROUP II |
|---------|-----------------------------|--------------------|---------------------|
| 20-29 | 5 | 12 | 17 |
| 30-34 | 8 | 13 | 14 |
| 35-39 | 25 | 13 | 16 |
| 40-44 | 14 | 16 | 8 |
| 45-49 | 19 | 13 | 15 |
| 50-59 | 17 | 22 | 19 |
| Over 60 | 12 | 11 | 11 |

thus compiled show that in the cholecystectomy group there had been about six times as many appendicectomies as in the control groups. Peptic ulcers were about twice as frequent in the cholecystectomy patients.

Evidence of rectal varicose disease was found in a surprisingly large number of the cases. Among the cholecystectomy patients no less than 79 had rectal varices. Examination of Control Group I emphasized the widespread existence of this complaint, for rectal varices were present in 43. In Control Group II there were 28 such cases. To sum up, the proportion of patients with rectal varicose disease was two or three times as great in the cholecystectomy cases as in the controls. About the same relation obtained for constipation. As many as 28 per cent of the cholecystectomy patients had had at one time or another anal fissures. In 14 cases, fissures in the acute stage were found, though this was not noted in the history. In the control groups, on the other hand, 8 healed fissures only were found. In addition there were 9 fistula cases among the cholecystectomy patients. The results are tabulated in *Table 3*.

Table 3—SHOWING INCIDENCE OF VARIOUS MORBID CONDITIONS IN CHOLECYSTECTOMY PATIENTS AND TWO CONTROL GROUPS

| HISTORY AND EXAMINATION | CHOLECYSTECTOMY CASES | CONTROL GROUP I | CONTROL GROUP II |
|-------------------------|-----------------------|-----------------|------------------|
| Appendicectomy | 31 (25) | 5 | 0 |
| Peptic ulcer | 10 (10) | 5 | 0 |
| Dysentery | 5 (5) | 7 | 0 |
| Typhoid | 88 (6) | 9 | 0 |
| Constipation | 71 | 30 | 0 |
| Blood in stool | 74 | 37 | 0 |
| Hæmorrhoids | 79 | 43 | 28 |
| Anal fissure | 28 | 8 | 0 |
| Fistula | 9 | 3 | 0 |

The figures in parentheses in the first column refer to the number of cases that had at the same time symptoms of rectal varicose disease (e.g., of the 31 appendicectomies 25 had some such symptom).

An interesting fact revealed by the statistics is that all of the 100 cholecystectomy patients had had some breach of the intestinal mucosa. Of this same group, no less than 37 showed evidence of either acute or chronic anorectal thrombophlebitis. Since our three groups differ in age and sex distribution, the values given by Hoff and Tietze for the incidence of these various diseases in a standard population group were taken, and the probable errors of the difference

between their figures and our cholecystectomy figures calculated Exhaustive examination of these data is impossible in the limited space of this book. We content ourselves with showing in *Table 4* the ratio of the actual differences found to the probable errors of difference

Table 4—SHOWING INCIDENCE OF VARIOUS CONDITIONS
IN CHOLECYSTECTOMY PATIENTS

| | | |
|---------------------|---|--|
| Appendicectomy | — | 5.5 times as great as probable error of difference |
| Peptic ulcer | | 1.1 |
| Dysentery | — | 0.3 |
| Constipation | | 5.2 |
| Blood in the stool | = | 5.2 |
| External noduli | | 5.3 |
| Fissure and fistula | | 4.3 |

It is in general agreed that a difference more than three times as great as the probable error of difference is statistically significant and this would apply to quite a number of the items, in some of which the proportion is well above this level. This holds for appendicectomy, constipation, blood in the stool, external noduli, and fissure and fistula, so that the incidence of each of these in patients who have had a cholecystectomy performed is significantly above the average. On the other hand the ratio for peptic ulcer is not significant (only 1.1 to 1), and the same may be said of dysentery.

From these figures it must be accepted that infection entering by the hæmorrhoidal plexuses plays an important part in the genesis of biliary disease. With regard to the route by which infection travels, we hold the view that organisms entering through a perforating appendicitis, through typhoid and paratyphoid ulcers and those of dysentery or cholera, or through anal fissures and fistulæ, as well as in all probability through the lesions of ulcerative colitis, pass to the liver in the portal blood stream.

This investigation may be said to confirm the very old belief that a relationship exists between 'piles', gastro-intestinal disease and biliary and liver disease. No gall-bladder patient therefore has been properly investigated until he has been thoroughly questioned and examined as to anorectal lesions. Hitherto as a rule no attention has been paid to this aspect of the cases. The converse is also true, that all patients with varicose rectal disease should be interrogated and examined for evidence of gastro-intestinal and biliary disease. We would again emphasize how striking it is to observe the disappearance of biliary attacks or dyspepsias simultaneously with the healing of the rectal lesions.

CHAPTER XII

ANORECTAL MUCOSAL PROLAPSE

It will be recalled that earlier in this book it was stated that digital examination of the rectum in patients with rectal varicose disease reveals important differences in the sphincter tone. While in some cases the muscle is firmly contracted against the examining finger, in others the sphincter action seems almost non-existent. In general, the longer the duration of the disease, the flabbier the sphincters and the pelvic floor musculature. We believe it to be the venous stasis and congestion which produces first a hypertonus and then a loss of tone of these muscles, similar reactions of muscle to congestion are met with elsewhere in the body, as already described. The hypertonic stage may be observed in almost all early fistulæ and fissures, but it passes in the course of time on to a weakness of the sphincters. The phenomenon may be compared with the tendency to ready fatigue found with varicose veins, which is probably the expression of a progressive impairment of the muscular power. The same applies to the bladder and the bowel, and the rule may perhaps be found to extend to the entire musculature, the heart, and the blood vessels. It is this behaviour of the muscles in the presence of prolonged venous congestion which is fundamentally responsible for anorectal prolapse.

Writers on the subject of prolapse have been impressed, perhaps unduly, by the fact that anorectal prolapse is often found associated with visceroptosis, varicose veins, flat feet, and utero-vaginal prolapse. This had led to the conception of a constitutional weakness of the connective tissue of the body as an aetiological factor. But it may be argued from the fact that varicose veins, rectal varices, and anorectal prolapse all tend to occur during pregnancy, that the same venous congestion may be the main cause of all of them. In our view, both rectal and uterine prolapse can be attributed to pelvic venous congestion. While in men the results are seen in the rectal prolapse, in women the uterus is the first to suffer. In women rectal prolapse is of rarer occurrence, but the two forms of prolapse are fundamentally due to the same causes.

Many of those patients who later develop prolapse of the anal mucosa complain at the beginning of a feeling of something in the rectum—a false defæcation impulse. This tenesmus is due to



Fig. 38. An. mucous membrane showing proctitis change in profuse secretion

increased sphincter tone sooner or later succeeded by a loss of the normal tone. Concurrently the chronic venous congestion and the flooding of the mucosal vessels with portal blood give rise to a proctitis, the weeping and œdematous condition of the mucosa corresponding to the eczema seen in the anal skin. The watery discharge from the mucosa induces a frequent desire to evacuate though no stool is present in the rectum (Fig. 38). The constant straining helps to produce a weakening of the levator ani muscles

and the whole pelvic floor. The patient believing himself to be constipated resorts to aperients—the favourite being a drastic saline



Fig. 39.—Hypertrophic folds of anal skin and mucosal prolapse

purge. Commonly the prolapse first appears after such a purgation. As a rule the prolapse is replaceable at first but unfortunately it becomes progressively worse until it interferes with the patient's work, and makes him afraid to go about freely. Bleeding and discharge become superadded and the undergarments require constant changing. Such patients may often be observed to stand with the legs crossed, hoping in this way to keep back the prolapse. Frequently the condition develops rapidly in patients with piles, after some strong aperient has been taken, and the sufferer does not



Fig. 40—Anal prolapse

realize that the prolapse should be replaced at once. Acute œdema of the prolapsed mucosa then develops and incarceration and necrosis may follow. As a rule even in these advanced cases replacement is possible with care. It is important, however, not to make the mistake of attempting also to reduce œdematous external noduli (*Figs 39, 40, 41*).

Many operations have been advised in the attempt to relieve this manifestation of varicose disease. Unfortunately these procedures are on the whole just as unsatisfactory as are operations for uterine prolapse. These measures depend on purely mechanical principles

and take no account of the aetiology of the condition. The primary and essential cause of the prolapse is the atrophy of the muscles of the pelvic floor due to venous congestion and portal backflow. This statement is supported by the successes we have had in the treatment of rectal prolapse by the vein compression method. The mucosal prolapse must be regarded as the last link in the chain of manifestations of the rectal varicose syndrome. Treatment should begin with the prevention by injections of the backflow of portal blood. This is carried out exactly as in the treatment of hæmorrhoids. In difficult cases the region of the sphincter itself may



Fig. 4.—Prolapse of anal mucosa

require to be injected in addition. This may be done two or three times under local anaesthesia.

The operative removal of strips of mucous membrane for prolapse has the same disadvantages as Whitehead's operation for piles. Often the bleeding and constipation are not relieved and cicatricial contraction or recurrence of the prolapse is all too frequent a sequela (Fig. 42). A large proportion of our cases of prolapse had already been operated upon unsuccessfully—often more than once. That these failures cannot be due to bad technique is suggested by the fact that even the most experienced rectal surgeons have a large

percentage of failures. Since prolapse generally occurs late in life, the cases suitable for operation are necessarily few. The patients and their relatives are generally slow to consent to the risks of an operation.

In the 'vein compression' treatment we have a method with which no surgical procedure can compete, and surgeons would be well advised to learn the details of the technique. This applies even to those who still prefer to treat hæmorrhoids by operation. More than 360 cases of prolapse have now been treated by the author with injections, and the strikingly successful results have

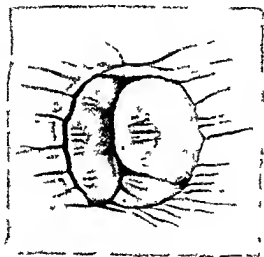


Fig. 42.—Prolapse after Whitehead's operation

only been equalled in the case of fissure. In 95 per cent there has been complete relief, and in the remainder great improvement. In fact, there have been no real failures in this series. There are, it is true, sometimes minor recurrences about a year later, necessitating further injections.

As a rule, prolapse needs more prolonged treatment than hæmorrhoids. Moreover, since the patients are usually older, some caution is needed. Not more than three deposits of 3 minims each should be injected at a sitting. Even with these small doses, improvement may be noted after the third treatment.

CHAPTER XIII

VARICOSE VEINS OF THE LEG IN RELATION
TO HÆMORRHOIDS

ALTHOUGH varicose veins are a very common complaint, their aetiology has never been satisfactorily worked out. It has been customary to account for their presence by some interference with the venous return—as for instance, valvular heart disease, abdominal tumours, or pregnancy. The enlargement of the uterus has always been considered to act as a mechanical obstruction. Yet, as has already been pointed out in Chapter VIII, the varicosities may develop at a stage when the uterine enlargement is quite insufficient to produce any mechanical effect. Consideration must be given to the alternative possibility that the same changes which produce the blue discoloration of the vulva (one of the first signs of pregnancy) may also be responsible for the development of varicose veins of the leg. We consider, as has already been discussed in Chapter VIII, that the reversal of stream which occurs during menstruation and pregnancy is one of the most important causes of pelvic congestion in women. It is therefore a logical step to ask whether these same factors may play a part in causing leg varices. Varicose veins, like anorectal varices, exist in a variety of forms. Thus, while in the thigh tensely filled tortuous veins as large as one's finger may be seen, at the ankle and on the dorsum of the foot the vessels are very small and no large ones are to be found. Similarly, the larger hæmorrhoidal dilatations lie above the sphincters, in the sphincteric part the vessels are smaller, and at the anal margin they are quite tiny.

Great stress has been placed upon the importance of the valves in the aetiology of varicose veins. In our opinion, valvular incompetence plays only a secondary role, in the superior hæmorrhoidal veins there are no valves, yet stream reversal only occurs in certain specified circumstances, as when the intra-abdominal pressure is raised during defæcation. It is quite probable that increase of intra-abdominal pressure is not the only factor which can disturb the circulation in this way. It may be that liver insufficiency, however caused, as well as sundry other factors can singly or together

disturb the balance between the portal and caval circulations. The following instances might be given —

1 The flattening of the diaphragm during defæcation may compress the inferior vena cava as it passes through this muscle

2 Chronic compression, displacement, or distortion of the caval opening (foramen quadratum) may be produced by hepatitis, cirrhosis, liver tumours or gummata, and similar conditions. Probably an enlargement of the liver may slightly compress the inferior vena cava in this situation, while any displacement of the heart might similarly distort the vessel with consequent embarrassment of the blood flow

3 An increased pressure in the main portal vein could lead to congestion in the ultimate branches of the hæmorrhoidal plexus, and through these pressure might be transmitted to the caval system

The close comparisons which can be made between the hæmorrhoidal and the saphenous venous systems have already been emphasized several times. No less striking than the anatomical and physiological comparisons are the pathological, thus —

| <i>Lower Limb</i> | <i>Rectum</i> |
|--------------------------------------|----------------------|
| Varicose ulcer | Anal fissure |
| Varicose erythema | Erythema ani |
| Varicose eczema | Anal eczema |
| Varicose pruritus | Pruritus ani |
| Thrombophlebitis | Peri anal thromboses |
| Acute suppurative thrombophlebitis | Peri anal abscess |
| Chronic suppurative thrombophlebitis | Anal fistula |

Just as varicose ulcers are usually found on the lower third of the leg, and hardly ever on the thigh, anal fissures are found at the lowest part of the anal canal. The same is true for erythema, eczema, and pruritus. Spontaneous thromboses occur in both rectum and lower limb, more commonly than anywhere else in the body. As has been previously explained, these thromboses are brought about by the backflow of portal blood into the caval vessels of the rectum and the leg respectively. On these lines, all the pathological manifestations can be explained, as well as the good results of injection treatment. It becomes clear also why no analogous conditions are seen in the upper limb. In the arm there is no such thing as varicose eczema, or localized pruritus. Neither ulcer nor thrombophlebitis occurs, and no varices of any importance are found. This is because there can be no connexion between the portal and the superior vena cava circulations. Since varicose veins of the leg are so often complicated by an ulcer, it is at first difficult to understand why ulcers

do not occur on the thigh, even though the veins may be varicose. Perhaps the explanation is that where numerous large collateral vessels exist, the portal toxins tend to be rapidly diluted and carried along while in the very small veins, the toxins are brought into such close relation to the skin that the latter is damaged. A similar argument may be applied to the rectum.

The tags of skin about the anus may be compared with the new formation of subcutaneous connective tissue in the leg. The bleeding from a hæmorrhoid resembles that from a ruptured varicose vein in the leg. The atrophy of the sphincter muscles may be compared with that which takes place in the calf. In so far as flat foot is the result of ligamentous and muscular weakening produced by varicose veins, it is comparable with anorectal prolapse, which results from rectal varices. Even the boring and stabbing pains complained of by patients with varicose veins are exactly similar to those experienced by sufferers from piles. Indeed, there is no manifestation of varicose veins which has not its counterpart in the rectal syndrome. Enormous varicosities may exist in the rectum without producing any symptom, while on the other hand, scarcely visible dilatations of minute veins may produce severe attacks of pain. In the same way, very large varicosities on the leg may be quite symptomless, while in cases where the objective signs are very slight, symptoms may be very severe.

There is no doubt that, in women, menstruation and pregnancy play the chief part in the causation of varices, whether of the rectum or the lower limb. The development of the varices will be favoured by each physiological reversal of the portal blood stream. In women, too, many auxiliary factors such as adnexal inflammation—acute or chronic œdema of the broad ligaments (parametritis)—may press upon the lateral pelvic wall and thus interfere with the flow of blood in the internal iliac vein. The same applies to ovarian tumours and uterine fibroids. In the male, of course, these factors are absent and the disturbances are chiefly central in origin. In the first rank is liver damage due to alcohol or syphilis. Cirrhosis and gummata are more frequent in the male than in the female. Hard physical labour and cardiac strain must also be considered. In brief, all those factors mentioned when discussing the ætiology of piles are equally important in the causation of varicose veins in men. Varicose veins and hæmorrhoids are, in other words, the outward manifestations of a more deep-seated complaint.

CHAPTER XIV

INSTRUMENTARIUM

Proctoscopes.—As long as twenty-five years ago, Foges, who had performed six thousand proctoscopies, gave his views on the diagnosis of internal hæmorrhoids. He emphasized that these could not be felt, but must be seen. The proctoscopes in general use then, as now, were quite unsuited to the diagnosis of higher-lying varices, as Foges well knew.

To overcome the disadvantages of such instruments, a series of proctoscopes with lateral windows or lateral slits was evolved during the early years of this century. Two instruments invented by Fergusson (*Fig 43*) are representative. Many modifications of the type were subsequently tried out.

Adaptations have been made and used by Abright and Kelly, Bensaude, Junghanns, Kirschen Blond, and Schur, among others. All these instruments are derived from a proctoscope produced twenty-five years ago in Vienna, an example of which has been presented to me. None of the instruments in use to-day can claim to be original, and this proctoscope, with its built-in electric light, compares very favourably with any in general use to-day (*Fig 44*).

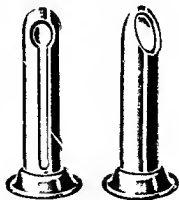


Fig 43—Fergusson's proctoscopes

Many of the modern proctoscopes, especially those with sliding apertures, are less efficient than this original model. In many, for example, if a polyp is trapped in the opening, the removal of the instrument without actually tearing off the polyp can be most difficult. We have devised a series of proctoscopes, of which the latest is a distinct improvement in several respects on older instruments. It is very compact and gives a view of the mucosa of a clarity never before attained. The instrument consists of a tube ending in a solid domed tip. Inside the tube is a polished metal mirror, and the

end of the tube is completely closed, which has the great advantage of keeping the proctoscope free from faecal matter. The size of the side window through which the rectal wall is examined has been so



Fig. 44.—Proctoscope produced in Vienna twenty-five years ago.

chosen that should a hæmorrhoid protrude into the lumen it may be disengaged with ease by rotating the tube. The instruments with sliding windows are defective in this respect. The light is controlled by a switch on the handle in which the battery is enclosed in a watertight compartment. Thus damage to the contents from water is completely prevented. The actual proctoscope tube is interchangeable and is quickly connected with the handle and light by a single milled screw. When attached, it may be rotated in either direction. For higher examinations the tube may be quickly replaced by a rectoscope.

A lens mounted on a pivot at the side enables the finest details, such as internal fistulous openings and small ulcers, to be examined. It is a most practical and efficient instrument of great general utility (Fig. 45).

Syringes—The drop-syringe is a very important item. There are quite a number of such instruments on the market. The ideal syringe for heavy duty should have the following points: (1) It must remain sterile without repeated boiling. (2) It must be always ready for instant use, to avoid delays. (3) It must deliver exact dosages. (4) The piston must not come in contact with the quinine solution.

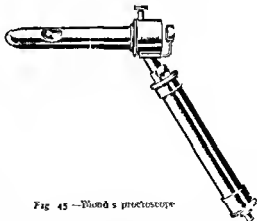


Fig. 45.—Biond's proctoscope.

The author has now produced a satisfactory syringe on these lines, and its design may be understood more clearly from the figure (Fig. 46) than from a verbal description. The injection solution is

supplied ready sterilized in a glass tube closed at each end with a rubber plug. One of these plugs is capable of being forced along

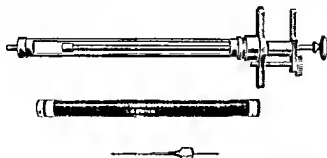


Fig 46—Blond's phial drop syringe showing the components

the tube by the piston of the syringe, after the whole tube has been placed in the frame of the latter. The needle, which is pointed at both ends, pierces the rubber plug at the other end of the tube, and is held in place by a screw fitting. A soft metal guard ensures that the needle does not penetrate more than a quarter of an inch or so into the tube. An additional advantage is that the needle thus attached cannot become loose or fall off into the rectum. The syringe may be used repeatedly, only the needle needing sterilization, because the piston does not come into contact with the solution. If some twenty to thirty needles are available there need be no delay. If the syringe falls, only the tube is broken and this may be replaced at once. If the contents are not used up on one case, the rest can be given to the next patient. The contents always remain sterile, only the needle needs to be changed.

Another useful syringe is that made by Melzer. It has been modified by Blond to overcome certain defects and includes a mechanism which delivers measured minim doses (Fig 47). The mechanism may be attached to an ordinary glass barrel like that of a tuberculin syringe. The piston-rod is notched and the notches engage with a lever (Fig 47 B). When the syringe is to be filled, this lever is disengaged by pressing the button (A). To eject the contents, the lever is pressed, and this

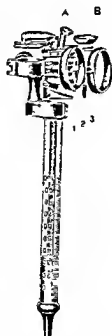


Fig 47—The Blond-Melzer drop syringe

forces down the piston, delivering a dose of 1, 2, or 3 minims according to the position of a regulator. The latter is adjusted by pressing it sideways, and moving it to one of three positions marked respectively 1, 2, and 3. It is held firm by a small stop which clicks into a hole in the regulator. Thus the descent of the piston is limited, and it is impossible to give more than the desired dose.

For injecting the solution into fistulæ, a blunt needle is used, as otherwise the wall of the track may be pierced and the solution placed in the surrounding tissue. For the paste, a special syringe is used, fitted with a screw-on cannula. It is illustrated in Fig. 31.

The complete equipment for treating fistulæ should include soft flexible probes, and a set of long sharp spoons. For very long, resistant fistulæ with rigid walls, a probe covered with insulating material as far as the olive end, which thus forms a diathermy electrode, is very useful.

Diathermy Apparatus.—We make extensive use of the diathermy for the removal of external skin tags and polypi, and for laying open short fistulous tracks. The apparatus which we use is specially adapted for this work.

To secure the best possible cutting current with a minimum effect

on surrounding tissue or cause coagulation, the wave-length is extremely short. Moreover, the voltage can be increased to give a 'flat' cut which heals well with very little reaction. By lowering the voltage and the frequency, a coagulating current is obtained for purposes of hæmostasis. The change over is carried out by simply pressing a button. Hæmostasis is most easily secured by using an artery forceps as an electrode, or with the spherical electrode (Fig. 48, 4), and the rapid change to coagulating current is most useful. The instrument is capable of very fine adjustment, and there is no faradic current. The accessories shown in Fig. 48 include

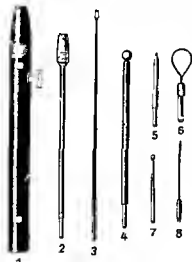


Fig. 48.—Diathermy instrumentarium.

all that is needed for the treatment of out-patients. The patient either lies or rests on the lead-plate electrode, it being unnecessary to remove the clothes.

The following accessories are illustrated in *Fig 48* (1) A handle with a finger-contact is used to hold the various electrodes. For working through the speculum an extension handle (2) is attached. The interchangeable wire snare (6), the needle (8), and the scalpel (5) are used for cutting. For coagulating fistulæ the probe-electrode (3) is used. The two ball-electrodes (4, 7) are also for coagulation purposes.

The Author's Polypus Forceps—This is an instrument designed for the removal of polypi. Their removal has previously required general or local anæsthesia and has often been accompanied by much

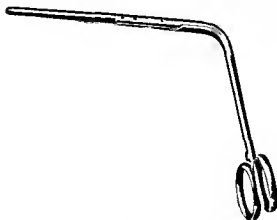


Fig 49—Blond's insulated polypus forceps

bleeding. Our improved technique consists in substituting a vulcanite tube for the usual metal one of the proctoscope. It has a metal ring upon it which is completely insulated. The polyp protrudes through the side window and a diathermy loop is slipped over it. It is then seized with the right angled insulated clamp (*Fig 49*) and the current is passed through the loop. The handle of the latter is gently pulled, and the polyp and clamp come away. The operation is simple, bleeding is minimal, and the entire procedure may be carried out in the out patient clinic without the need for any anæsthesia or special preparation.

REFERENCES AND BIBLIOGRAPHY

- ATCHULDI, A., "À propos du Traitement des Hémorroïdes par la Méthode sclérosante Un Cas d'Intolérance aigue par le Chlorhydrate double de Quinine et d'Urée (Kinurea)", *Presse méd.*, 1930, 38, 1355
- ADLER, "Palliative Treatment of Anal Fissure", *Matthieu's Med Quart.*, 1894, 1, 383
- ALBUT, GUY, THÉBAUT, and DA COSTA, "Au Sujet du Début des Lésions d'Hépatite et de Cholecystite au cours d'une Infection générale", *Bull Soc med Hôp de Paris*, 1931, series 3, 47, 1313-19
- ALLINGHAM, *Pruritus Ani in Diseases of the Rectum and Anus*, 1888, 198.
- ANDERSON, H. G., "After-results of Operative Treatment of Hæmorrhoids", *Brit Med Jour.*, 1909, 2, 1276-9
- ANDERSON, H. G., and DUKES C., "The Treatment of Hæmorrhoids by Submucous Injections of Chemicals" *Ibid.*, 1924, 2, 100-2
- ANDERSON, H. G., "The Injection Method for the Treatment of Hæmorrhoids" *Practitioner*, 1924, 113, 399-409 (bibliography)
- BARTHOLDY, K., *Arch f klin Chir* 1902, 66, 956-76
- BAUMANN and BLOND, "Zur Aetiologie und Therapie der sogenannten Fistula Ani", *Schweiz med Woch* 1936, 17, 358-61
- BALL, SIR CHARLES *The Rectum, its Diseases and Developmental Defects*, reviewed in *Zentralbl f Chir.*, 1909, 36, 116
- BELLOT V. J., "Traitement sclérosant en une séance des Hémorroïdes Résultats d'une Expérience de cinq Ans", *Arch de Med et Pharm nat.*, 1928, 118, 1237
- BENDA, *Handbuch für Pathologie*, chapter "Gefässe"
- BENSAUDE, R., and OLRY, P., "Traitement des Hémorroïdes par les Injections sclérosantes sept Ans d'Expérience", *Presse méd.*, 1928, 36, 706
- BLOND, K., "Zur Gastritisfrage" *Beitr z klin Chir.*, 1931, 152, 578-80
- BLOND, K., "Die Beziehungen des sogenannten Circulus vitiosus nich Gastro enterostomose zum Ulcus pepticum jejuni postoperativum", *Arch f klin Chir.*, 1925, 135, 281-339
- BLOND, K., "Ein weiterer Beitrag zur Genese der postoperativen Jejunal geschwüre", *Ibid.*, 1927, 144, 245-65
- BLOND, K., "Eine neue Arbeitshypothese zur Klärung der Gallenwegsprobleme" *Ibid.* 1928, 149, 662-99
- BLOND, K., "Ein experimenteller Beitrag zur aufsteigenden Invagination in den operierten Magen", *Ibid.*, 1928, 153, 53-61
- BLOND, K., "Ueber Duodenektomie und eine neue Methode der Transplantation des Ductus choledochus und pankreaticus", *Ibid.*, 1930, 156, 736-57
- BLOND, K., "Wandlungen in der Lehre von der Funktion der Gallenwege" *Ibid.*, 1932, 170, 597-647
- BLOND, K., "Postoperative Komplikationen in der Magen Chirurgie", *Der Chirurg*, 1929, 1, 845-9
- BLOND, K., "Cholezystographie und Funktion der Gallenblase", *Fortschr a d Geb d Röntgenstrahlen*, 1930, 41, 571-81
- BLOND, K., "Zum Entleerungsmechanismus der extrahepatischen Gallenwege", *Deut med Woch*, 1928, 54, 778-9
- BLOND, K., "Ueber ein bisher nicht beachtetes Symptom bei der Cholezystographie und seine klinische Bedeutung", *Klin Woch*, 1929, 8, 1572-3
- BLOND, K., "Zur Frage des mechanischen Ikterus", *Wien klin Woch*, 1932, 45, 1348-9

- BLOND, K., "Zur Gallenblasenphysiologie und Pathologie", *Klin Woch.*, 1927, 6, 1606-8
- BLOND, K., "Die Verödungstherapie des Hämorrhoidalleidens", *Der Österreichische Arzt*, 1935, Nos 3, 4, also *Gesellschaft der Ärzte, Wien*, Protokoll vom 8 Juni, 1934, and *Wien klin Woch.*, 1934, 24
- BLOND, K., "Zur Pathologie und Therapie des varikösen Symptomenkomplexes des Mastdarms", *Wien klin Woch.*, 1934, 47, 1409-12
- BLOND, K., "Die Spontanthrombose des Plexus hämorrhoidalis inferior", *Med klin*, 1935, 31, 880-3
- BOAS, J., "Obstipation und Hämorrhoiden", *Zentralb f ärztliche Fortbildung*, 1916, also *Das Hämorrhoidalleiden*, 1922, Halle, Carl Marhold, and *Deut med Woch*, 1927, 1929
- BODENHAMMER, *Treatise on Aetiology, Pathology, Symptoms, and Treatment of Anal Fissure*, 1868
- BOHM, C., "Beiträge zur ärztlichen Praxis", 1937, 6/9, *Med Klin*, 1938
- BORCHARD, "Chirurgie des Mastdarms und Afters", in Garré, Kuttner, and Lexer's *Handbuch*, 3 (bibliography)
- BOYER, *Traité des Maladies chirurgiques*, 1894, 494, 605, Paris
- CAMPANOCCHI, "Sekundäre durch trockene pleuropulmonale Tuberkulose hervorgerufene Cholezystopathien", 43 *Kongress der deutschen Gesellschaft für innere Medizin* 1931, 315
- CHIARI, "Ueber die analen Divertikel und ihre Beziehungen zu den Anal fisteln", *Medizinische Jahrbücher, Gesellschaft der Ärzte, Vienna*, 1878, 419
- CELSUS, cited from Greffrath
- COPELAND, *Dublin Hosp Gaz*, 1855
- COUDRAY, G., "Traitement des Varices et des Hémorroïdes au moyen des Injections sclérosantes"
- DELATER and VENDEL, "A propos de la Fibrose curative des Hémorroïdes par Injections", *Presse med*, 1928, 36, 1329
- DUNBAR, J., "The Treatment of Hemorrhoids by Interstitial Injections", *Brit Med Jour*, 1923, 2, 808
- DÉONDI, cited from Greffrath
- EDWARDS, F SWINFORD, "The Treatment of Piles by Injection", *Brit Med Jour*, 1888, 2, 815-16
- EDWARDS, F SWINFORD (jointly) "The Injection Treatment of Hemorrhoids" *Practitioner*, 1915, 94 343-57
- ELSNER, H., "Die Behandlung der Hämorrhoiden mit Alkoholinjektionen", *Deut med Woch*, 1927, 657, and 1932, 1324, *Münch med Woch*, 1932 2, 1174
- EPPINGER, *Asthma cardiale*, 1924 Springer
- ESMARCH, *Die Krankheiten des Mastdarmes und des Afters*, 1887 127 Enke, Stuttgart
- FAGAN, E P *Surg Clin of N Amer*, 1933, 13, 1337
- FERGUSON, "Treatment of Recto vaginal Fistula" *Matthew's Med Quart*, 1895, 2, 157
- FISCHER, A W., "Verwachsungen der Gallenblasenerkrankungen Zur Pathologie der doppelten Gallenblase", 26 *Tagung der süddeutschen Chirurgen Breslau (Gießen)*
- FOGES, *Atlas der rektalen Endoskopie*, 1909, Urban and Schwarzenberg, Berlin and Vienna, also *Wien klin Woch.*, 1918, 31, 360
- FRANKEL, *Deut med Woch* 1932, 2, 1487
- FRERICHS and LANCERAUX, *Handbuch für Dermatologie*
- GANT, *Die Krankheiten des Mastdarmes und des Afters*, 1904, 1, Munich
- GLASER, A, *Deut med Woch*, 1931, 785, *Med Klinik*, 1931, 1245., *Med Welt*, 1932, 1, 599
- GOZ, E., "Kasuistische Beiträge zur Fistula ani", *Beitr z klin Chir*, 1916 99/2, 268-93
- GRAEFE and ULZER, review of literature up to 1904
- GREFFRATH, "Beitrage zur Operation der Mastdarmlisteln", *Deut Zeits f Chir*, 1887, 26, 18

- HABER, *Lekar Rev*, 1938, Prague
- HACKER, "Excision des ungespaltenen Ganges der ischiorektalen Fistel und primäre Naht behufs Vermeidung der Inkontinenz", *Zeits f Chir*, 1919, 858
- HARTMANN, "De la tuberculose anale", *Rev. de Chir*, 1894, 4
- HINRICHSSEN, H M, "Betrachtungen über die Zusammenhänge zwischen Appendicitis, Cholecystitis, und Ulcus ventriculi und Ulcus duodeni", *Beitr z klin Chir*, 1927, 140, 149
- HIRSCHMANN, L J, *Handbook of Diseases of the Rectum*, 4th ed, 1926, 173, London
- VON HOCHENEGG, *Lehrbuch der Chirurgie*, 1908, Urban und Schwarzenberg Berlin and Vienna.
- HOOTON, cited from Bensaude and Oury
- HOWARD, C, *Lancet*, 1929, 1, 20, 1931, 914 (pruritus ani)
- JUNGHAANS, *Arch f klin Chir*, 1933, 178 2 (recent bibliography)
- KELLY, H A, "New Method of Examination and Treatment of Diseases of the Rectum and Sigmoid", *Ann of Surg*, 1895 21, 468-78
- KELSEY and LANGE, in Garré, Küttner, and Lexer's *Handbuch*, 7, 959
- KILBOURNE, N S, *Ann of Surg*, 1934 99, 600
- KIRSCHEN, *Wien med Woch*, 1932, 1576, *Wien klin Woch*, 1934 19
- KIRSCHNER NORDMANN, *Handbuch der Chirurgie*, 1930, 5, Urban und Schwarzenberg Berlin and Vienna
- KONIG, *Lehrbuch der speziellen Chirurgie*, 1889, 2, 432, Berlin, *Arch f klin Chir*, 70 (peri anal abscess fistula formation)
- VON LANGENBECK cited from Garré, Küttner, and Lexer's *Handbuch* 1923
- LAUDA, ERNST, "Die Behandlung der Erkrankungen der Leber und Gallenwege", *Med Klinik*, 30, No 39 1
- LERICHE, RENÉ "Ueber die Forschungsmethoden in der Chirurgie", *Deut Zeits f Chir*, 1934 243, 489
- LESER, *Spezielle Chirurgie*, 3rd ed, 1897, 447, Jena
- LOCKHART MUMFERY, J P, and JOSHI, M K, *Lancet*, 1915, 2, 322
- LOCKHART MUMFERY, J P, *Diseases of the Rectum*, 1923 Baillière Tindall and Cox, London
- MAISON NEUVE, *Clinique chirurgicale*, 1864 2 Paris
- MARESCHE, *Pathologische Anatomie Behandlung des Rektums und Sigmas*, 168
- MARINO, A W M, *Amer Jour Surg*, 1934 48 23, 366-70
- MARTIN, F, cited from Bensaude and Oury
- MEISEL, P, *Beitr z klin Chir*, 1900 28, 293-302
- MEISEN, W, *Acta chir Scand*, 1928, 64 311, 1931, 58, 14
- MELCHOR, E, "Ueber die Wirkung der einmaligen Durchtrennung des Afterschliessmuskels", *Beitr z klin Chir*, 1910, 70, 745 (fistula) 1916 104, 436 (division of sphincter) also *Munch med Woch*, 1910 1989 *Berliner klin Woch*, 1917, 621 (tuberculosis)
- MEYER, K, "Die Bakteriologie der Gallenerkrankungen und ihre Bedeutung für die Pathogenese", *Klin Woch* 1933, 12 73
- MITCHELL A B, "A Simple Method of Operating on Piles", *Brit Med Jour* 1903, 1, 482
- MORGAN C NAUNTON, "Oil-soluble Anesthetics in Rectal Surgery", *Ibid*, 1935, 2 938
- MORLEY, A S, *Hæmorrhoids Their Aetiology Prophylaxis and Treatment by Means of Injections*, 1924 London, also *Lancet*, 1928, 1 543
- MOZKOWITZ, "Ueber Periproktitis und Fistula Ani", *Arch f klin Chir*, 1920 114, 745, also *Wien klin Woch*, 1925, 38, 177
- MOYNIHAN, B, Mitchell Banks Memorial Lecture, "On the Gall bladder and its Infections", *Brit Med Jour*, 1928, 1 1
- NOTENAGEL *Spezielle Pathologie und Therapie*, 1896, 17, Vienna
- PARÉ, AMBROISE, *The Works of Paré* English version by Johnson, 1854 83 954, London
- PELLER, *Wien Arch f inn Med*, 1922, 4, also "Der funktionelle Zustand der peripheren Gefässe", *Wien klin Woch*, 1923, 10

- PILLA and BILLROTH, 3/2a, 133
- POSSELT, "Beziehungen zwischen Leber, Gallenwege, und Infektionskrankheiten", *Ergebn d allg Path u path Anat*, 1927, 22, 1, 590
- QUÉNU and HARTMANN, "Fistules Ano rectales", in *Chirurgie du Rectum*, 1895, 158, Paris
- RÉCAMIER, "Massage Cadencé", *Rev med de Paris*, 1898, Jan
- REICHLE TIETZE, *Handbuch der Chirurgie* by Kirschner and Nordmann, 5 (bibliography)
- RICHTER, W., *Med Welt*, 1932, 1174
- RITTER, "Zur operativen Behandlung von Mastdarmfisteln, die oberhalb des Sphinkter in den Darm münden", *Zentralb f Chir*, 1920, 92
- ROTTER, *Handbuch der praktischen Chirurgie*, 1927, 5, *Arch f klin Chir*, 1900, 61, 866
- VON RYDQVIST, *Deut Zeits f Chir*, 1908, 492
- SACKS, G., *Brit Med Jour*, 1933, 1, 313
- SENDLER, *Handbuch der Chirurgie*, 1927, 5
- SMITH and LANGE, in Gerre, Küttner, and Lexer's *Handbuch*, 1886, 930
- SCHARF, S., "Appendektomie als aetiologischer Faktor der Gallenblaserkrankungen", *Med Klinik*, 1934, 1125
- SCHIEFFELAR, *Ref Zentralorgan f Chir*, 1925, 33 593
- SCHLAGER, L., *Lekar Rev*, 1938
- SCHREIBER, *Rekto romanoskopie*, 1903, Hirschwald, Berlin
- SCHLHARDT, "Über die tuberkulöse Mastdarmfistel", *Volkmann's Sammlung klin Vortr*, 1887, 296
- STADLER H., *Schweiz med Woch*, 1931, 1098, Basle
- STIEFF, W., "Krankheiten der Verdauungsorgane", in *Lehrbuch der inneren Medizin*, v Bergmann, 1931, 750
- STERNBERG, "Behandlung der Mastdarmfisteln", *Zentralb f d ges Ther*, 1903, 129-97
- STRAUSS, *Prokto sigmoidoskopie*, 1910, Thieme, Leipzig
- STROHMMEYER, *Handbuch der Chirurgie*, 1851, 2, 636, Freiburg
- TAVEL, "Cystische Entartung des Sinus Hermann und Genese der Analfisteln", *Deut Zeits f Chir*, 1902, 62, 399
- TAVEL and MEISEL, in Gerre, Küttner, and Lexer's *Handbuch*, 3
- TERRELL, E H., *Amer Jour Surg*, 1921, 35 382
- VILARDELL, J., "Zur Pathogenese der Cholecystitis (die Rolle der Hepatitis)", *Arch f Verdauungskrankh*, 1932, 51, 207
- WEISS, *Munch med Woch*, 1932, 346
- WHITEHEAD, *Lancet* 1881, 2, 112
- WILDE, *Med Klinik*, 1937
- WUNDERLICH, *Handbuch der Pathologie und Therapie*, 1856
- ZAUN, *Dermatol Woch*, 1933, 2, 1320-4

| | PAGE | | PAGE |
|---|----------|--|-------------|
| * HABITUAL constipation venous stasis and | 52 | Ligature method in anal fistula | 101 |
| Hæmorrhage after pelvic operations | 66 | Liver affects us in aetiology of hæmorrhoids | 3 |
| — — treating hæmorrhoids statistics | 23 | — — eclampsia | 70 |
| — from internal varices | 9 11 | — — pruritus and | 68 |
| Hæmorrhoidal papilla proctoscopic appearance (Fig 4) | 13 14 | MARITAL peri anal abscess | 79 |
| — plexus inferior pruritus and | 56 | — thrombophlebitis chronic (see Anal Fissure) | |
| — veins thrombophlebitis of (see Thrombophlebitis) | | Melzer drop syringe Blond s modification | 131 |
| Hæmorrhoids aetiology of | 1 5 | Menopause pruritus at (Fig 47) | 67 |
| — diagnosis and investigation of (Figs 1 5) | 10-15 | Menstruation pruritus and | 67 |
| — internal in anal fissure | 38 39 50 | Metabolites in portal blood pruritus and | 56 60 61 67 |
| — symptomatology | 6-10 | Methylene blue to locate inner opening of anal fistula | 105 |
| — thrombosed (see Nodulus) | | Micturition portal stasis affecting | 51 |
| — treatment of general (Figs 6 7) | 16-25 | Morgagni sinuses of fistula formation and | 98 |
| — technique (Figs 8 9) | 25 37 | Mortality after injection treatment of hæmorrhoids case reports | 29 |
| Healing of fissures and fistulae 88 et seq | | — — operative treatment of hæmorrhoids statistical | 23 |
| Heart disease in aetiology of hæmorrhoids | 3 | Mucocutaneous junction thrombosis of varices at | 6 |
| Hermann sinuses of fistulae and | 95 | NECROSIS in injection therapy of hæmorrhoids | 25 9 34 |
| Historical survey of treatment of anal fistulae | 99 | Needle diathermy (Fig 48 (5)) | 13 |
| History taking in hæmorrhoids | 11 | Nitric acid in treatment of anal fistula | 100 |
| Horseshoe fistula in ano (Fig 21) | 85 | Nodulus external in cholecystectomy patients | 120 |
| Hydrogen peroxide to locate inner opening of anal fistulae | 105 | — terminology | 2 |
| Hypertonus of sphincter ani in anal fissure | 40 46 | — thrombosed evacuation of clot (Figs 6 7) 18 (Fig 8) 25 | |
| — — — internal varices | 8 | OPERATIVE treatment of anal fistula | 103 |
| Hypotonus of sphincter ani in internal varices | 9 | — — hæmorrhoids arguments for | 22 |
| Idiopathic hæmorrhoids aetiology of | 4 | — — — criticism of statistics | 32 |
| Incarceration of prolapsed hæmorrhoids | 15 123 | — — — statistical | 43 |
| Incomplete fistulae | 86 | — — — old recurrent fistulae case histories of failures (Figs 32 36) | 107 |
| Inflammatory theory of hæmorrhoids | 2 5 | PAIN in anal fissure | 40 42 44 46 |
| Injection treatment in anal fissure (Fig 12) | 48 | — due to internal varices | 8 11 |
| — — — fistula (Fig 31) | 104 | Pancreatic tumours in aetiology of hæmorrhoids | 3 |
| — — — case histories (Fig 37) | 111 | Papilla hæmorrhoidal proctoscopic appearance (Fig 4) | 13 14 |
| — — — following operative failure (Figs 32 36) | 107 | Pelvic operations incidence of embolism after | 65 |
| — — anorectal mucosal prolapse | 125 | Pelvicorectal abscess | 79 |
| — — habitual constipation | 53 | Pelvis female veins of (Fig 17) | 71 |
| — — hæmorrhoids | 16 23 | — male veins of (Fig 14) | 62 |
| — — — statistical | 23 | Peptic ulcers in aetiology of gall bladder disease | 119 |
| — — — technique (Fig 9) | 26 | Peri anal abscess | 7 11 78 |
| — — pruritus | 73 | — — classification | 86 |
| — — suppurative thrombophlebitis | 81 | — fistula (see Fistula in ano) | |
| Instrumentarium (Figs 43 49) | 129-133 | Peripræpitis | 78 |
| Inspection in anal fissure | 44 | — following injection treatment in anal fissure | 49 |
| — hæmorrhoids | 12 | Phenol solutions in injection treatment of hæmorrhoids | 16 23 |
| Intramural peri anal abscess | 79 | Pile sentinel in anal fissure | 38 44 |
| Ischio-rectal abscess | 79 | — terminology | 2 |
| Itching peri anal | 12 | Plantar pain in anal fissure | 44 |
| — in pruritus ani | 66 | | |
| JALDICE pruritus and | 67 68 | | |
| KIDNEY right tumour of in aetiology of hæmorrhoids | 3 | | |
| LANGENBECK'S operation advantages of injection therapy over | 21 | | |
| Laxatives in anal fissure | 47 | | |
| Leg varicose veins of in relation to hæmorrhoids 1 9 14 58 64 126-128 | | | |

| | PAGE | | PAGE |
|--|--------------|---|----------------------|
| Polypus forceps, Blond's use with diathermy (Fig 49) | 133 | Stercoral ulcers | 84 |
| Portal circulation blood stasis in, in aetiology of internal varices | 3 15 | Stone in common bile-duct in aetiology of haemorrhoids | 3 |
| — — — — causes of | 68 | Structure following treatment of haemorrhoids (statistical) | 23 |
| — — — — constipation in | 51 | Subcutaneous abscess | 86 |
| — — — — pruritus and | 54 | Submucosal injections in haemorrhoids | 77 |
| Pregnancy pruritus and | 67 69 | Submucous abscess | 86 |
| Probe electrode diathermy (Fig 48 (3)) | 132 | Suppurating piles | 83 84 |
| Proctitis chronic | 63 | Suppurative thrombophlebitis (Figs 18 30) | 78-98 |
| Proctoscopes (Figs 43 45) | 129 | — — following injections for fissure | 49 |
| — injection of haemorrhoids via (Fig 9) | 26 | Symptomatology of haemorrhoids | 6-10 |
| Proctoscopy in haemorrhoids (Figs 1-5) | 12 | Syphilitic anal fistulae and ulcers (Figs 27 28) | 94 |
| Prolapse anorectal mucosal (Figs 38 42) | 121 125 | Syringes for injection therapy (Figs 46 47) | 130 |
| — of internal varix | 9 11 15 | — quinine paste injections (Fig 31) | 106 |
| Prostatectomy bleeding after | 61 | Syringotomy in excision of anal fistula | 100 |
| — in senile pruritus | 67 72 | | |
| Pruritus and in fissure | 42 | TERMINOLOGY | 1 2 |
| — — haemorrhoids and | 54 | Thrombophlebitis acute anal | 7 8 11 78 |
| — — injection treatment of | 73 | — — — classification | 86 |
| — — local and general (Figs 13 17) | 54 | — aetiological classification | 81 |
| Pudendal plexus post partum changes | 77 | — in aetiology of anal fissure | 39 43 et seq |
| Pus in anal fissure | 41 | — — biliary disease | 117 120 |
| | | — chronic anorectal (see fistula in ano) | |
| QUININE paste injections in anal fistula (Fig 31) | 106 | — suppurative (Figs 28 30) | 78-98 |
| — rash after injection treatment of haemorrhoids | 29 33 et seq | — — after injections for fissure | 42 |
| Quinine urea solutions in injection treatment of haemorrhoids | 16 23 28 | Thrombosed varices external treatment of (Figs 6 7 18 (Fig 8) | 25 |
| | | — — at mucocutaneous junction | 6 |
| RACE haemorrhoids and | 4 | — — suppurating | 83 84 |
| Radiography in fistula in ano (Figs 19 20) 87 (Figs 23-26) | 39 | Thrombosis of rectal veins spontaneous | 54 |
| Rash quinine after injection treatment of haemorrhoids | 29 33 | Treatment of anal fissure (Fig 12) | 47 |
| Reactions in injection therapy of haemorrhoids | 28 et seq 33 | — fistula in ano (Figs 31 37) 99 116 | |
| Rectal veins spontaneous thrombosis of | 54 | — haemorrhoids general (Figs 67) 16 25 | |
| Rectum and anus acute and chronic suppurative thrombophlebitis of (Figs 18 30) | 78-98 | — — technique (Fig 8 3) 25 37 | |
| — blood supply of (Fig 13) | 59 | Trendelenburg's sign | 3 9 58 |
| | | Tuberculous perianal fistula | 93 97 |
| SAPHENOUS system varicosities in anal, to haemorrhoids | 9 14 58 64 | Tumours pancreatic in aetiology of haemorrhoids | 3 |
| Scalpel for diathermy (Fig 48 (5)) | 132 | — renal in aetiology of haemorrhoids | 3 |
| Sedentary occupations haemorrhoids and | 5 | ULCER of anus varicose (see Anal Fissure) | |
| Sentinel pile in anal fissure | 38 44 | — peptic in aetiology of gall bladder disease | 111 |
| Sigmoid cancer rectal varices in | 12 | — stercoral | 84 |
| Sign Trendelenburg's | 3 9 58 | Uterus venous drainage of (Fig 16) | 11 |
| Sinus(es) of Hermann fistula and Morgagni fistula and | 98 | | |
| Skin changes in pruritus and (Fig 15) | 66 | VEINS of female pelvis (Fig 17) | 71 |
| Skin tags production of | 7 | — male pelvis (Fig 14) | 62 |
| — termination | 2 | — rectum (Fig 13) | 59 |
| — treatment of | 25 | — — spontaneous thrombosis of | 54 |
| Sloughing following injection of haemorrhoids (statistical) | 23 | — uterus (Fig 16) | 69 |
| Sphincter and dilatation or division of in anal fissure | 47 | Venous impression therapy (see Injection Treatment) | |
| — — division of fistula | 80 | Varices (see Haemorrhoids) | |
| — — hypertonus of in anal fissure | 40 | Varicose ulcer of anus (see Anal Fissure) | |
| — — internal varices | 8 | — veins of leg in relation to haemorrhoids | 3 9 14 58 64 126-128 |
| — — hypotonus of in internal varices | 9 | | |
| Spontaneous thrombosis of rectal veins | 54 | WHITEHEAD'S (perianal) advantages of injection therapy | 21 |
| | | — — — after (Fig 47) | 124 |
| | | Wire snare diathermy (Fig 48 (1)) | 132 |